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BLOOD GROUPS IN GILBERTESE.

By J. J. GRAYDON, R. T. SIMMONS and N. M. SEMPLE,
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AND

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IN a discussion of the results of an early survey of blood group frequencies in Maoris (Graydon *et alii*, 1946) reference was made to the lack of similar data in Micronesia and in the borderline island groups which might have served as intermediate resting places for the Polynesians on their journey to their present Pacific homeland. At that time there was some published work on the A-B-O blood groups in natives of the Carolines, but nothing was known of the blood group frequencies to be found in the inhabitants of the Gilbert and Ellice Islands, which may well have figured in the prehistory of the Polynesian.

The islands of the Gilbert group consist of atolls composed almost entirely of coral rising only about eight feet above sea level. Vegetation is very limited, being of coconuts and pandanus. Ocean Island, on the far western boundary of the Gilbert group, is composed of phosphate and coral rock, and is more fertile. The islands are of two kinds; some have a lagoon in the middle and are geologically older than others which have a lagoon open to the sea. The sixteen islands have a population density of 600 to the square mile, the versatile coconut providing in large measure the necessities of life.

The Gilbertese are a hardy, virile people of good physique, and unlike most other tropical island natives, they work hard. This is no doubt due to the high population density and the necessity to fish vigorously and skilfully to provide sufficient food. They are intelligent, well-behaved, moral and polite. Although nominally Christians, they still carry a wholesome respect for magic. They are nearly always happy and see the funny side of everything. In physiognomy they are different from the Polynesians, from whom they may be readily distinguished even by casual observation.

The post-war resumption of operations by the British Phosphate Commission on Ocean Island necessitated the indenture of native labour from nearby sources. It was thought that opportunities would present themselves on Ocean Island to obtain representative samplings from the natives of the Gilbert and Ellice Islands and possibly also the Marshall Islands.

When a number of Banabans (who, though natives of Ocean Island, live in the Fiji group) returned to settle some questions of land boundaries, advantage was taken of their presence on Ocean Island to collect blood samples from them. Blood was collected only from those who

TABLE I.
Blood Phenotypes in Gilbertese.

Lot.	Subjects.	A-B-O Groups.				M-N Types.			Rh Types.				Number Tested.
		O	A_1	B	A_1B	M	MN	N	Rh_1Rh_1	Rh_1Rh_2	Rh_2	Rh_1Rh_2	
I	Banabans (Ocean Island)	4	12	17	9	10	18	14	17	7	6	12	42
II	Gilbertese (random)	13	46	21	9	15	34	40	43	9	4	33	89
III	Gilbertese (random)	3	12	8	5	2	11	15	16	1	4	7	28
	Total Gilbertese	20 (12·6%)	70 (44·0%)	46 (28·9%)	23 (14·5%)	27 (17·0%)	63 (39·6%)	69 (43·4%)	76 (47·8%)	17 (10·7%)	14 (8·8%)	52 (32·7%)	159

stated that their parents and grandparents were Banabans. This means that in this group (Lot 1 of this survey) there is no admixture with other Gilbertese blood, and when the age of these subjects (averaging about fifty years) is also considered, there seems no likelihood of there being any European or foreign admixture, as Europeans did not settle on Ocean Island until the early 1900's. These specimens of blood were stored at 34° to 40° F. for a few days after collection on November 27, 1947, and thence shipped in the cool room (40° F.) of the motor vessel *Trienza* to Melbourne, where they were tested twenty days after collection. The samples arrived in perfect condition, showed no signs of hæmolysis and gave satisfactory results with all sera used for testing.

The second group from which samples were collected in March, 1948, was a more or less random sampling of Gilbertese consisting of hospital attendants, nurses, patients and others from the neighbourhood of the hospital on Ocean Island. The samples were sent back to Melbourne in the same way as the first lot, and the results of tests performed within fifteen days of collection were satisfactory on 89 of the 90 samples collected. Only one sample had to be discarded because of hæmolysis.

The third lot of 28 samples was collected in March, 1948, from a similar group of Gilbertese; but owing to lack of suitable transport they were not received in Melbourne until September 28, 1948. They were then six monts old, but this was not known in the laboratory at the time of testing. During the interval the samples had been stored in a refrigerator maintained at a temperature between 34° and 40° F. They were shipped to Melbourne in the cool room of the motor vessel *Triona*. There was no hæmolysis in any of the samples on receipt and all tests gave satisfactory results.

Eleven of the sixteen islands were represented in the second and third lots, which comprised samples from individuals born on Abaiang (11), Arorae (10), Beru (20), Maiana (3), Marakei (5), Nikunau (14), Nonouti (12), Onotoa (13), Tabiteuea (20), Tamana (8) and Tarawa (1).

TABLE II.
Blood Gene Frequencies in Various Populations of the Western Pacific Area.

Population.	Authors.	Number of Samples Tested.	A-B-O*			M-N		Rh				
			A	*B*	*O*	*m*	*n*	R^0	R^1	R^2	R^z	*r'*
Gilbertese	Present survey	159	0·382	0·278	0·340	0·389	0·611	0·076	0·691	0·233	—	—
Marshall Islanders	Simmons, Graydon *et alii* (1952)	678	0·136	0·135	0·729	0·222	0·778	0·0065	0·9516	0·0419	—	—
Maoris	Graydon, Simmons *et alii* (1946)	447	0·347	0·003	0·650	0·503	0·497	0·027	0·465	0·486	—	0·022
	Simmons, Graydon *et alii* (1951)	180										
Papuans	Simmons, Graydon and Woods (1946)	455	0·187	0·149	0·664	0·133	0·867	0·021	0·943	0·020	0·016	—
		100										
Admiralty Islanders	Simmons and Graydon (1947)	112	0·146	0·136	0·718	0·339	0·661	0·030	0·940	0·030	—	—
Fijians	Simmons, Graydon and Barnes (1945)	310	0·206	0·106	0·688	0·326	0·674					
	Simmons and Graydon (1947)	110						0·11	0·84	0·05	—	—
Javanese	Simmons and Graydon (1951)	262	0·178	0·180	0·642	0·632	0·368	0·065	0·837	0·086	0·012	—
		205										
Other Indonesians (20 islands).	Simmons and Graydon (1951)	239	0·130	0·133	0·737	0·435	0·565					
	Simmons and Graydon (1947)	100						0·04	0·81	0·13	0·02	—
Natives of Borneo	Graydon, Simmons *et alii* (1952)	584	0·211	0·196	0·593	0·542	0·458	0·039	0·873	0·079	0·009	—
		580										
Filipinos	Simmons and Graydon (1945)	382	0·148	0·178	0·674	0·510	0·490	0·03	0·88	0·08	0·01	—
		100										

* The A-B-O gene frequencies given in this table have been calculated from the original data by Fisher's method (Race and Sanger, 1950) and may differ slightly from those presented in the original papers; also, in some cases, data obtained in separate surveys have been pooled to give closer approximation to average values for the relevant populations.

The samples were collected by finger puncture after thorough cleansing of the skin with ether soap followed by methylated spirits and a final ether swabbing. Two to four drops of blood were taken into vials containing one millilitre of sterile glucose-citrate solution (Simmons and Graydon, 1945).

Results and Discussion.

The results of tests for the A-B-O, M-N and Rh types have been summarized in Table I, in which the three series are shown separately.

In Table II gene frequencies calculated from these results have been compared with those of neighbouring and possibly related populations, which have been examined by the present authors with various collaborators.

The Gilbertese possess an exceptionally high frequency of gene *A* (0·382), which is not approached by any other population studied to date in Indonesia, Melanesia and Micronesia with the exception of the Trukese, in whom the *A* gene frequency is 0·315 (Simmons, Graydon and Semple, in the press). A high *A* frequency is apparently the rule in Polynesia, for Nigg (1930) obtained a value of 0·380 for *A* in Hawaiians, and the present authors in two separate surveys obtained values of 0·352 and 0·338 for the frequency of the *A* gene in Maoris. It may be argued that the high *A* frequency of the Gilbertese links them closely with Polynesians; but this view is difficult to reconcile with the findings for the *B* gene, which in Gilbertese reaches the highest value (0·278) yet reported for Melanesia, Micronesia and Indonesia, in contrast to its virtual absence in Polynesia. The exceptionally high values for the *A* and *B* genes combine to make the frequency of *O* in Gilbertese lower than in any other Pacific island group.

Although it is not unlikely that random genetic drift has been at some stage an important factor in determining gene frequencies of these native peoples, it would seem necessary to look further afield than nearby island groups for some of the racial elements which, by hybridization, could produce such unusual A-B-O frequencies as those observed in Gilbertese.

The racial composition of the Micronesians was discussed by Birdsell in a recent paper (Simmons *et alii*, 1952) and he suggests that a Caucasoid component of Ainoid origin has contributed appreciably to the genetic complex of this region. It is interesting to note that in one area of Hidaka, North Japan, Simmons *et alii* (1953) found in 112 Ainu an A frequency of 0·261, a B frequency of 0·360 and an O frequency of 0·366, which figures differ little from the corresponding values in Gilbertese.

In the present series there were 70 samples of group A and 23 of group AB, all of which belonged to sub-groups A_1 and A_1B respectively. The absence of the sub-group A_2 is typical of native populations inhabiting all Pacific coastlines and islands.

The M-N frequencies of the Gilbertese are intermediate between those found in the peoples of New Guinea and the Admiralty and Marshall Islands on the one hand, and those of the Polynesians and Indonesians on the other. They are also similar to M-N values reported for the group of Ainu previously mentioned.

As anti-S serum was not available at the time when this survey was made, no data were obtained for the S subdivisions of M-N types. It is hoped that some future opportunity will present itself to test Gilbertese blood samples for this and for other factors which have since assumed great anthropological importance.

Only four Rh phenotypes were found in the Gilbertese, and these may be accounted for satisfactorily by assuming the presence of three "genes", R^0(cDe) or *r*(cde), R^1(CDe) and R^2(cDE). The gene R^z(CDE), which has been proved to exist in several of the Western Pacific populations, and gene *r'*(Cde), which was reported in Maoris, were not found. The frequencies of genes R^1(CDe) and R^2(cDE) in Gilbertese are intermediate between those found in Polynesia (Maoris) and those found in Melanesia, Indonesia and the Marshall Islands. The distribution of Rh types in Gilbertese is again somewhat similar to that in the Ainu group previously referred to, but there is one striking difference: the gene *r''*(cdE), common in Ainu, was not found in Gilbertese. This gene was found once in a Javanese survey (Simmons and Graydon, 1951) associated with *r'*(Cde), and therefore may have come south, but been generally swamped by other major components.

The evidence provided by this study has revealed no clear-cut pointer to the hybridization represented in Gilbertese. One may suggest that a strong Polynesian element has been superimposed on a population differing from those groups now found in Melanesia and Micronesia (with the possible exception of the Trukese), in that it has been more strongly influenced by an Ainoid component.

Of the racial elements which may be represented in Gilbertese we can say little, except to point out that hybridization of Negritoid, Ainoid and some Mongoloid elements with a later superimposed Polynesian group could have produced blood gene frequencies fairly close to those observed in the present-day Gilbertese.

The appreciable Polynesian influence indicated by the blood grouping data for the Gilbertese could be interpreted as support for the widely accepted theory that the Polynesians reached their present-day homeland via the Micronesian route, passing through the Gilbert Islands on the way. However, the Gilbert and Ellice Islands may have received their Polynesian immigrants by westward migration from Polynesia proper, and the absence of group B in Polynesia and other blood grouping data tend to favour this hypothesis.

Summary.

1. A total of 159 Gilbertese blood samples taken from natives of Ocean Island and 11 other islands of the Gilbert group were tested for A-B-O, M-N and Rh types.

2. The results were as follows: O, 20 samples (12·6%); A, 70 (44·0%); B, 46 (28·9%); AB, 23 (14·5%); M, 27 17·0%); MN, 63 (39·6%); N, 69 (43·4%); Rh_1Rh_1, 76 (47·8%); Rh_1Rh_2, 17 (10·7%); Rh_2, 14 (8·8%); Rh_1Rh_2, 52 (32·7%). The subgroups A_2 and A_2B were not found.

3. Gene frequencies calculated from these results were as follows: *A*, 0·382; *B*, 0·278; *O*, 0·340; *m*, 0·389; *n*, 0·611; R^0, 0·076; R^1, 0·691; R^2, 0·233.

4. The racial composition of Gilbertese is briefly discussed.

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THE SOCIOLOGICAL PROBLEM OF HEMIPLEGIA.[1]

By W. S. DAVIDSON,
Deputy Commissioner of Public Health, Western Australia.

A GLANCE at Table I (taken from Linford, 1951) refutes the suggestion frequently made that the productive age groups are bearing an ever-increasing burden because of an aging population. The growth of the aged group is counterbalanced by the decrease in the non-productive group of the earlier years of life. The proportion of the productive years group to the whole has been increasing throughout the period shown. The reduction in young age groups *plus* the efforts of preventive medicine and more recently chemotherapy and the antibiotics has rendered the infectious disease hospital obsolescent—many are half empty or turned over to other purposes. Despite this reduction in the burden we bear in looking after the sick and handicapped, we complain bitterly of the ever-increasing proportion of the population who, because they are aged, require more and more hospital and institutional care. It would seem fitting that having dealt effectively with the problem of the sick child and lightened the demands upon the community from the sick in this group, we should now turn some of our enthusiasm and resources to dealing with the growing problem of the aged sick. The main purpose of this paper is to indicate that this enthusiasm and effort have been woefully lacking and that a better appreciation is required, both by the medical profession and by society in general, of what can be done to alleviate the silent suffering of the aged sick.

It is well nigh impossible to deal with the subject fully in the space of one paper. The plight of the hemiplegic has therefore been taken as an example. The position of the hemiplegic patient may be considered indicative in greater or lesser degree of many other diseases of the elderly or aged.

In July, 1950 (Davidson, 1950), a survey of all the chronic sick in Western Australia was undertaken to ascertain the size of the problem and the extent to which it might be alleviated. A chronic case was defined as that of a person who in our present state of medical knowledge was likely to require in-patient treatment for the remainder of his life or who would require frequent readmission to hospital for relapses of the same disease. Pulmonary tuberculosis and mental disease or mental deterioration were not included in the group.

Reports were obtained from all chronic hospitals, institutions and boarding houses dealing with such cases, and all patients more than eight weeks in acute hospitals were similarly examined. To obtain a figure for those patients unable to obtain admission to hospital, 22 doctor groups submitted reports on "chronics" in their practices. These practices covered 85,000 people or one-sixth of the total population.

The survey indicated that throughout the State our requirement for chronic cases was 2·1 beds per 1000 of population. This distribution was not even; a larger proportion was required in the metropolitan area than in rural areas. Our figure of 2·1 closely approximates the United States of America estimate of 2·0 per 1000 and the United Kingdom estimate of 2·5 per 1000. An estimate was similarly prepared of cases in which the patient did not require hospital accommodation but required admission to an institution. The figure arrived at for institutional cases was 1·4 per 1000 of the population.

The main groups of diseases represented in these chronic and institutional cases as they occurred in 1087 cases in the metropolitan area are shown in Table II.

Hospitals and institutions throughout the State reported on 724 patients classified as chronic and 435 classified as suitable for an institution. Doctors covering one-sixth of the population reported on 79 chronic cases and 46 institutional cases in which the patients were unable to obtain admission. If we multiply the doctors' cases by five we are not over-estimating the total cases in the State in which admission was not effected. We therefore find that there are in the State 1121 chronic cases and 665 cases in which the patients are suitable for institutional care.

Among the 725 chronic cases in hospital *et cetera* were 72 cases of hemiplegia, and in the 435 institutional cases were 24 of hemiplegia.

TABLE I.
Population Age Distribution, Australia, 1871–1947.

Year of Census.	Persons (Percentage).			
	Under 15 Years.	15 Years and Under 65 Years.	65 Years and Over.	Total.
1871	42·09	56·17	1·74	100
1881	38·89	58·61	2·50	100
1891	36·92	60·19	2·89	100
1901	35·14	60·88	3·98	100
1911	31·65	64·08	4·27	100
1921	31·71	63·86	4·43	100
1933	27·48	66·04	6·48	100
1947	25·05	66·90	8·05	100

In the 79 chronic cases reported by the doctors were six of hemiplegia, and in the 46 institutional cases were five of hemiplegia. If we multiply the doctors' cases by five, as before, we estimate the number in the whole population in which patients were unable to obtain admission to hospital or institute. The position is then shown in Table III.

This table shows a total of 151 cases of hemiplegia in which the patient required hospital or institutional care existing at one time in a population of 500,000. That is, for every 100,000 of population, there are 30 hemiplegics requiring hospital *et cetera* care at an annual cost of approximately £20,000. This does not include cases in which financial and family circumstances are such that the patients may be treated in their own homes at their own expense.

TABLE IIa.
Main Disease Groups, Chronic and Long-Stay Cases.

Total Number of Cases in Group.	Age Group. (Years.)	Main Diseases.	Number of Cases.
56	0 to 14	Post-poliomyelitis	15
		Diseases of bones and joints	11
		Deformities	11
		Rheumatic fever	7
		Nephritis	4
163	15 to 59	Fractured femur	19
		Arthritis	12
		Hemiplegia	10
		Other fractures	8
		Paralysis agitans	8
		Post-poliomyelitis	7
		Cardiac disease	6
433	60 and over	Cardiac disease	72
		Hemiplegia	54
		Senility	53
		Malignant growth	33
		Arthritis	32
		Fractured femur	15
		Prostatic enlargement	14

The age distribution of the 107 reported cases is given in Table IV, and the diagnosis in the reports are listed in Table V.

As these are all chronic cases, it is reasonable to infer that the majority are the result of cerebral thrombosis, although apparently the diagnosis is rarely ascertained beyond the level of hemiplegia. Cerebral and subarachnoid

[1] Read at a meeting of the State Health Council of Western Australia on April 12, 1953.

hæmorrhage we must surmise will account for most of the remainder.

The resulting paralysis from these lesions represents nearly 9·1% of the demand on our "chronic" beds and 7·4% of institutional accommodation.

Tables VI (A) and VI (B) give some details of 28 male and 17 female hemiplegic patients in "chronic" hospitals. These are not selected patients but the total hemiplegic population of the hospitals examined. It is to be noted that

TABLE IIB.

Main Diseases in Cases for Institutional Care.

Total Number of Cases in Group.	Age Group. (Years.)	Main Diseases.	Number of Cases.
16	15 to 59	Cardiac disease	3
		Hemiplegia	2
		Brain tumour	2
419	60 and over	Senility	127
		Cardiac disease	76
		Arteriosclerosis	39
		Arthritis	27
		Hypertension	24
		Hemiplegia	23
		Asthma, chronic bronchitis	23
		Fractured femur	5

the disabling paralysis occurred not at the first but at the second or third attack in no less than 14 of the 45 cases and that the interval between the strokes varied from days to years. The most important fact displayed is the length of time for which these patients have been paralysed. The time varies from two months to twenty-three years. The average in both male and female patients is over three years. This, however, gives little indication of the expectation of life in the hemiplegic, as, although the average is between three and four years, the variation around the mean is so extreme that no mean can give a

TABLE III.

Hemiplegia Among Chronic and Institutional Patients.

State.	Chronic Patients.			Institutional Patients.		
	Total.	Hemiplegics.	Percentage.	Total.	Hemiplegics.	Percentage.
In hospital *et cetera*	726	72	—	435	24	—
Unable to be admitted	395	30	—	230	25	—
Total	1121	102	9·1	665	49	7·4

true picture. Furthermore, if the duration of life in all hemiplegic cases was taken, the period would be considerably reduced by the large number of patients who die within hours of the initial stroke. Our figures do not even indicate the expectation of life in the chronic case, because all these people were alive when interviewed, and many of them were very much alive. The figures can only indicate that the hemiplegic patient can and frequently does live for many years. In other words, the cardio-vascular catastrophe that initiates hemiplegia is not necessarily the beginning of the end of life but merely the beginning of a long period of torment and despair. Once we appreciate this, the inadequacy and inhumanity of our present attitude and efforts on behalf of the hemiplegic become apparent.

What may be done and what can be done are ascertained from a consideration of prevention, treatment and rehabilitation.

Prevention and Treatment.

There is at present no certain method of predetermining the onset of a cerebral thrombosis, but a considerable amount of work is being done on factors influencing the onset of coronary artery thrombosis and atherosclerosis. When these are determined, we hope in the not too distant future, it is reasonable to suppose that, *ipso facto*, we will have a means of predetermining the onset of thrombosis, be it coronary or cerebral. Our planning, then, must include the possibility of such prognostication.

Preventive treatment, however, need not wholly await the unravelling of the mysteries of thrombosis. As shown, no less than 14 patients out of 45 developed their hemi-

TABLE IV.

Number in Various Age Groups of 107 Reported Cases of Hemiplegia.

Year of Decade.	Number of Patients in Age Groups (Years).					
	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 to 89
0	—	—	—	2	8	3
1	—	—	—	1	8	4
2	—	—	—	4	3	1
3	—	—	1	6	5	3
4	—	1	—	4	4	2
5	1	1	1	3	2	1
6	—	1	—	7	7	2
7	1	—	—	1	1	—
8	—	—	2	4	4	1
9	—	—	—	2	4	1
Total in decade	2	3	4	34	46	18

plegia, not on the first attack, but on a subsequent one. Close questioning indicated that in several other cases there were premonitory signs from a few hours to a few days before the actual stroke.

It is therefore relevant to consider the possibility of antithrombotic treatment in such cases to prevent the onset of the stroke or of the subsequent stroke that converts a minor paresis into a major paralysis.

Rose (1950) writes: "There is a striking lack of published information concerning the use of anticoagulants in cerebral infarction. This is most likely due to the fact that any patients so treated showed such a poor response

TABLE V.

Diagnoses Recorded in 107 Reported Cases of Hemiplegia.

Diagnosis.	Number of Cases.
Hemiplegia	60
Stroke	11
Cerebral thrombosis	8
Hemiplegia with cardiac lesion	6
Hemiplegia with arteriosclerosis	6
Cerebral hæmorrhage	5
Hemiplegia with hypertension	3
Hemiplegia with diabetes	1
Hemiplegia with stricture	1
Hemiplegia with infantile paralysis	1
Hemiplegia with mental defect	1
Hemiplegia with cancer of bowel	1
Hemiplegia with Parkinson's disease	1
Hemiplegia with spastic effects	1
Cerebral diplegia	1
Total	107

that it was not thought fit to continue the work." This may be true, but, in view of its vital importance to a large body of suffering humanity, it must be borne in mind that it is but an assumption the truth of which requires further demonstration.

In a series of 24 cases of hemiplegia, including three of "spasm" of cerebral arteries, Rose came to the conclusion that the use of anticoagulants was of no benefit in the functional result. He was, however, mainly concerned with

the treatment in the acute case in which the hemiplegia had developed, rather than with the prevention of the initial hemiplegia.

Scott (1952) claims good results in the prevention of coronary and cerebral thrombosis from long-continued low dosage of anticoagulants. The data he gives do not allow a full evaluation of the claims, but if substantiated they would give considerable hope of preventing subsequent severe attacks in cases in which the initial attack has produced only minor damage. They would also indicate a method of treatment to be used when we are in a position to predetermine the onset of such thrombosis.

In cerebral hæmorrhage the prognosis is more serious, and the vast majority of affected patients die within a few hours. Falconer (1952) indicates that even in cerebral hæmorrhage much can be usefully accomplished by active and skilled interference.

TABLE VIA.

Onset and Duration of Hemiplegia in 28 Males Still in Hospital.

Age at Time of Review. (Years.)	Incapacitation Produced by First, Second or Third Stroke.	Interval Between Strokes.	Duration of Hemiplegia.	Associated Disease where Recorded.
71	2	4 years.	9 months.	Hypertension.
67	1	—	1 year 2 months.	—
45	1	—	2½ years.	—
63	1	—	8 years.	—
84	1	—	1 year.	—
76	1	—	3 years.	—
72	2	10 days.	2 years.	—
55	1	—	1½ years.	—
70	1	—	1 year 3 months.	—
74	2	3 months.	1 year 2 months.	—
68	1	—	4 years.	—
70	1	—	2 years.	—
65	1+	—	7 years.	—
74	2	7 months.	1 year 7 months.	—
80	2	2 months.	9 months.	Hypertension.
71	2	1 month.	1 year.	—
52	1	—	4 months.	—
71	1	—	2 years.	—
71	1	—	9 months.	—
70	1	—	1 year 8 months.	—
73	1	—	6 years.	—
52	1	—	8 years.	—
84	1	—	1½ years.	—
70	2	1 year.	5½ years.	—
58	1	—	2 years.	—
66	1	—	11 months.	—
43	1	—	16 years.	—
46	2	2 weeks.	2 years.	—

Rehabilitation.

In the application of physical medicine to the case of the hemiplegic we are no longer speculating regarding what may be done in the realm of experimental medicine. We are dealing with what can be done, what has been done and what should invariably be done.

As an introduction to this part of the subject, I quote extracts from a private communication from Mr. M. J. Rosen:

> The physiotherapist is usually called upon to treat the patient long after the optimum time. It is typical in hemiplegics to find well-established contractures that could have been readily prevented, but are almost impossible to cure.
>
> The intensity of the treatment given the patient is also of prime importance in influencing their recovery. Except in the case of the wealthy patient private treatment is usually inadequate because the patient cannot afford the constant care that must be given. In actual practice we usually find the severe hemiplegic is doomed to spend his last days (usually many years) in the hopelessness of an institute for the chronic sick. This, to my mind, is the last place that any patient with any possible chance of rehabilitation should be relegated.
>
> In my own experience, I have found that once admitted to these places, treatment is made doubly difficult by the conviction of the patient that his condition is hopeless, otherwise he should not have been sent there. There is no provision made for physiotherapy treatment in these institutions as they were actually designed for those patients who could never return to normal life.
>
> This underlines the necessity of some means of treating patients who would require treatment for a considerable time, but still have the possibility of at least returning to their families, if not of leading an actual active life.
>
> As the general hospitals have not the beds to cope with this problem, special provision should be made for them.

The following two case reports illustrate the difference between early adequate treatment and late inadequate treatment:

CASE I.—Married woman, aged 60. Had stroke 10/4/49. Immediately admitted to hospital with complete paralysis on L. side including aphasia. Power of speech returned rapidly; but there was no voluntary movement when physiotherapy was started on 27/4/49. Although a difficult patient, judicious bullying got her out of bed and walking with assistance. A severe spastic inversion of the ankle was treated by a shaped leather anklet. Daily treatment was given until 15/9/49—then three times weekly and later once weekly. Her present condition is that although there is no voluntary movement in the foot, and only a little in the knee, she can walk aided only by a stick. Lives in a first floor flat and can negotiate stairs unaided. Voluntary movement in hand and arm is almost nil, but she does tapestry work and embroidery on a frame, reads, plays cards, etc. She goes to town twice weekly to attend to her business affairs; goes visiting, motoring and away for holidays. Still seen once weekly to make sure no contractures arise.

TABLE VIB.

Onset and Duration of Hemiplegia in 17 Females Still in Hospital.

Age at Time of Review. (Years.)	Incapacitation Produced by First, Second or Third Stroke.	Interval Between Strokes.	Duration of Hemiplegia.	Associated Disease where Recorded.
65	1	—	2 years.	—
80	2	3 weeks.	3 years.	—
70	1	—	3 years.	—
62	3	Total: 3 years.	1 year.	—
74	3	Total: 3 weeks.	1 year.	—
87	2	7 years.	6 months.	Diabetes.
77	1	—	1 year.	—
73	2	5 years.	14 years.	—
70	1	—	2 years.	—
75	1	—	9 years.	? Coronary attack one year before stroke.
69	1	—	2 months.	—
65	1	—	7 months.	—
49	1	—	5 months.	Hypertension.
69	1+	—	1½ years.	Hypertension.
74	2	?	23 years.	—
49	1	—	3½ years.	Mitral stenosis and embolism.

CASE II.—Male aged 69. Had stroke 25/9/51. Nursed at home and various hospitals until admitted chronic hospital 24/9/52. Originally had complete paralysis L. side. Physiotherapy commenced 15/1/53. At this stage, patient had excellent movement in all muscles. Very little spasm and only slight inco-ordination. There was a severe kyphosis because of a complete neglect of the patient's bed-lying position. (Head propped up; but not shoulders). The good voluntary movement would suggest that the patient would soon walk, but this is proving most difficult. This is mainly due to the terrible posture and complete loss of balance. This patient is being treated three times weekly; but requires much more intensive treatment plus a constant watch on his position in bed. He is most unhappy in his present environment and very emotional. I consider that this case could leave hospital in a comparatively short time if the economics of his treatment did not come into the picture.

The benefits bestowed by physiotherapy, the improvement and remarkable degree of rehabilitation that are possible in cases of hemiplegia from the patient and skilful application of physical medicine have been recorded by Dinken (1949), Lowman (1948), Rusk (1950), Howell (1948), Licht (1949), Covalt (1949) and others.

According to Krusen (1949), "one of the significant contributions in physical medicine and rehabilitation has been the demonstration of the considerable potentialities of proper physical treatment and retraining of hemiplegics. Every hemiplegic should be given the advantages of a well organised programme such as has been described by Dinken".

Lowman describes the result of physiotherapy in 18 late or chronic cases and in 10 early cases of hemiplegia. He writes:

> In 66% of the chronic cases reambulation was accomplished after an average period of 118½ days. Improvement had occurred in an additional 17 per cent. In the remaining 17 per cent. no benefits were obtained because of mental deterioration and emotional instability of a degree which precluded adequate co-operation.
>
> In 90% of the fresh cases reambulation was accomplished after an average period of 67·1 days. Improvement occurred in the remaining 10%. Among this group there were no failures.

These results are in conformity with the general favourable picture painted by other writers on the subject. Why then do our "chronic" hospitals house so many chronic hemiplegics whose only treatment is an occasional anodyne? The fault would seem to arise from two basic misconceptions: a mistaken medical appreciation and an ill-conceived economic evaluation. To quote Dinken:

> The general practitioner of medicine is primarily responsible for the care of this large group of hemiplegic patients. It is essential that he face this task with realistic optimism, for much can be done to help restore these persons to lives of independence and productivity.

There are four things that interfere with this "realistic optimism". Firstly, there is the lack of appreciation of the chronicity of the disablement or the length of time the patient lingers in misery. Secondly, there is a poor appreciation of the benefits that may be bestowed in the way of rehabilitation or partial rehabilitation by physiotherapy. Thirdly, a fear exists that active treatment may worsen the cerebral damage. In this latter respect Dinken may again be quoted:

> Treatment should be instituted as soon as possible. In the majority of cases it can be started within the first week following onset. There are few medical contraindications to early treatment in most cases, especially to passive movement of the involved extremities daily, which is particularly important. The fear of producing extension or reactivation of the cerebral vascular lesion is apparently without justification, as indicated by clinical experience.

Fourthly, the medical practitioner is restrained in his optimism by the hopelessness of obtaining a long and expensive course of treatment for a patient who no longer has an earning capacity.

This brings us to a consideration of the economics of diagnosis, prevention, treatment and rehabilitation. These require skilled techniques, expensive apparatus and prolonged application far beyond the financial means of most hemiplegic patients. The means must, therefore, be found by the State and can be obtained only in competition with other claims on the public purse. It must, however, be borne in mind that effective treatment will not only cause a tremendous saving in costly hospital beds but also restore the productive capacity in a greater or lesser degree of a large proportion of our hemiplegic population. Apart from economic values, civilized society cannot avoid its obligation to eradicate the inhuman neglect at present universally evident in our "chronic" wards and institutions, where adequate therapy would prevent the hopelessness and despair of the twisted, tortured, spastic hemiplegic and restore him to some degree of hope, movement and rehabilitation. The relief of the patient would be reflected many times in the joy of relatives and friends, who must at present watch, in helpless anguish, his years of suffering.

Conclusion.

The burden of unproductive age populations is no greater on us than it was on our forefathers, yet we bear it with an ill grace. Much of the money that is now being spent on little short of neglect toward sufferers from hemiplegia could more profitably be spent on special clinics, wards, equipment and skilled operators to prevent onset of the disease or to treat and rehabilitate its victims.

Such clinics and institutions would also function for the investigation and relief of diseases requiring similar equipment, such as coronary artery thrombosis and arthritis.

Summary.

Tables show that the productive portion of our population has not decreased in proportion to the non-productive.

The main diseases of the chronic sick are given.

Our requirement of "chronic beds" is 2·1 per 1000 of population. For institutional beds it is 1·4 per 1000 of population.

Hemiplegia accounts for 9·1% of the demand for "chronic" beds and 7·4% of the demand for institutional beds.

It is suggested that the care of the hemiplegic is woefully neglected, and much expense to the State and misery to the sufferer could be saved by a proper application of prevention and treatment.

The introduction of special clinics, wards *et cetera* for the care of hemiplegics is suggested.

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THE PROBLEM OF ENDEMIC GOITRE.

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ENDEMIC GOITRE is one of the most widespread and serious food deficiency diseases in Australia today, and large numbers of the populations in Papua and New Guinea are affected. Since surveys have not been made in all parts of Australia, it is possible that the condition is even more widespread than existing information suggests.

From surveys already made the following areas may be considered to be goitrous: (i) Queensland, Cairns and the Atherton Tableland (Sutton, 1927); (ii) New South Wales, large areas of the northern and southern tablelands, the Hunter Valley and the headwaters of the Namoi (Sutton, 1927; Clements, 1948); (iii) Victoria, Gippsland and scattered localized areas around Colac, Ballarat, Hamilton (Sutton, 1927; Clements, 1948); (iv) South Australia,

Adelaide Hills area (Jungfer, 1948); (v) Tasmania, most of the island (Clements, 1948); (vi) Papua and New Guinea, large areas of the highlands and foothills (Clements, 1936).

In December, 1952, the World Health Organization (WHO) convened a meeting to review various aspects of the problem of endemic goitre. This meeting, which was attended by doctors and scientists[1] interested in the subject, was held at the National Institute of Medical Research, Mill Hill, London. A report of the meeting by WHO will appear, in due course, in one of the official publications of the organization. The purpose of this paper is to relate the findings of the conference to the problem in Australia and her dependencies.

Ætiology.

The meeting accepted the following statement by the Goitre Subcommittee of the Medical Research Council of Great Britain:

> The immediate cause of simple goitre is failure of the thyroid gland to obtain a supply of iodine sufficient to maintain its normal structure and function. This failure is usually brought about by an absolute environmental deficiency of iodine; it may also be caused by factors which interfere with the availability of dietary iodine or which impose an abnormal demand on the thyroid gland.

The acceptance of this definition means the non-recognition of the so-called physiological goitre of adolescence. Enlargement of the thyroid gland in this age group indicates insufficient iodine in relation to demands.

The basic deficiency is in the iodine content of the soil in which vegetables and fruits are grown and on which milk cows are pastured. The iodine content of surface water is frequently a good index of the iodine content of the soil over which it runs. Many of the soils deficient in iodine have had the iodine and other soluble trace elements leeched out over long periods of time.

The commonest sources of iodine in a non-goitrous area are vegetables, especially green leafy vegetables, and milk, and these are followed by cereals and meat. Water contributes about 5% of the normal intake. Proximity to the sea does not ensure protection from goitre, except in that it may provide a liberal supply of fish. However, in Tasmania endemic goitre exists in fishing villages because the whole catch is sent to the Melbourne market. Contrary to popular beliefs, winds blowing off the sea contain infinitesimal quantities of iodine. Sea salt is an extremely poor source of iodine; the average concentration is one part per 10,000,000.

The Public Health Importance of the Problem.

Simple enlargement of the thyroid gland during childhood and adolescence frequently presents an æsthetic problem at this time.

A significant number of women who had an endemic goitre in adolescence experience during pregnancy further enlargement of the gland, which reveals an insufficient intake of iodine to meet the additional demands of the fœtus. In some women the enlargement is pronounced and is often associated with the development of nodules in the gland. This sequence is responsible for the troublesome enlargement in middle life, which at times leads to pressure effects so that recourse must be had to surgery. The London meeting noted that the presence of endemic goitre in an area is associated with an increase in the number of thyroid operations. Apart from the effects of these operations on the patients, they also represent an additional economic burden in terms of hospital costs.

[1] Those who participated were the following: Dr. E. Burgess, WHO, Geneva; Dr. L. E. Campbell, Food and Agriculture Organization, Rome; Dr. S. Gilder, WHO, Geneva; Mr. J. C. M. Holman, London; Dr. F. C. Kelly, London; Dr. O. P. Kimball, Cleveland, U.S.A.; Dr. J. Matovinonic, Zeboa, Yugoslavia; Professor M. Murray, London; Dr. J. Nicol, Lausanne, Switzerland; Dr. M. E. Pochin, London; Dr. A. Quirido, Leiden, Holland; Dr. U. Ramalingaswami, Coonoor, India; Dr. H. M. Sinclair, University of Oxford; Dr. J. B. Stanbury, Boston, U.S.A.; Dr. E. Stacpoole, Mexico City; Dr. Selwyn Taylor, London; Dr. B. T. Towery, Nashville, U.S.A.; Dr. F. W. Clements, Australia (who acted as chairman).

The relationship of endemic goitre to secondary toxic goitre has been the subject of several investigations. Wyndham (1940) studied a large group of patients with secondary toxic goitre who had been operated on in a Sydney hospital. One third of these had a preexisting endemic goitre. He also found that the highest incidence per 1000 of population throughout the State, excluding the patients from Sydney and Newcastle, was from the endemic goitre areas. This study confirmed observations made overseas and reported by Eggenberger (1938). The London meeting accepted the existence of this relationship, and emphasized the importance of prevention as a long-term step in the reduction of the incidence of secondary toxic goitre.

The possible relationship of carcinoma of the thyroid to endemic goitre was briefly discussed. The members found themselves unable to express an opinion, largely because of lack of information. Australia provides possibilities for the study of this aspect of the problem. Queensland and Western Australia seem, from data available, to have relatively few goitrous areas compared with Tasmania.

The classical studies in Switzerland demonstrated a close relationship between endemic goitre and cretinism. However, although the relationships between feeble-mindedness and deaf-mutism and endemic goitre are frequently accepted by some writers, the meeting did not think these had been clearly established. Recent studies have shown that in an average population a high percentage of the feeble-minded children are but the lower part of the normal distribution curve of intelligence and so represent the effects of multifactorial inheritance. This conclusion calls for new studies on this aspect of the problem, which probably does not affect the white population of Australia as much as native groups in the Pacific Islands.

The Prevention of Endemic Goitre.

Endemic goitre is preventible. It is, in fact, one of the easiest of the food deficiency diseases to prevent, as has been demonstrated in the United States of America (Kimball, 1949), in Switzerland (Eggenberger, 1938), in Mexico (Stacpoole, 1952) and in parts of Australia, as will be outlined below. With the exception of the Australian experiments and the Mexican work, the goitre has been prevented by the universal use, in the areas affected, of iodized salt. Influenced by these results, the English Parliament has just enacted legislation requiring domestic salt to be iodized. Owing to technical difficulties associated with the task of treating all salt, the law has not yet been promulgated.

The recent meeting in London recommended that in countries where endemic goitre occurs all food salt should be iodized. If it is assumed that the daily requirement for iodine is approximately 100 microgrammes and that the daily consumption of salt is about 10 grammes, the rate of iodization of food salt should be one in 100,000. When, for some reason, only table salt is iodized the rate should be one in 20,000. The meeting recommended that iodization should be legally mandatory.

Potassium iodide has been added to bread in a number of areas of Holland, and this has proved a satisfactory procedure, for it has been followed by a significant fall in the incidence of endemic goitre.

A form of iodine prophylaxis for endemic goitre has been in force in parts of Australia for about five years. The form used is the administration of a tablet containing 10 milligrammes of potassium iodide. The scheme started in Canberra in 1947 shortly after that city was found to be in a goitrous area. Later the scheme was extended to Tasmania and parts of Victoria.

So far as is known no scheme for the prevention of goitre other than the voluntary use by householders of iodized salt, when available, is in operation in New South Wales, South Australia or Queensland.

The Canberra project commenced as an experiment designed to test the effectiveness of this method of distribu-

tion, and to ascertain whether it was possible by this method to supply adequate quantities of iodine to infants and young children whose salt intake was negligible. The tablets were distributed to children at school and were available for infants and pre-school children through infant welfare clinics. The effects of this method of distribution of iodine on thyroid enlargement on one age group in Canberra is shown in Table I. I am indebted to Dr. Edith Clement for the figures for 1951 and 1952.

Equally striking results have been obtained after two and a half years of prophylaxis in most of the districts in Tasmania where this method has been used. The results from two districts are not nearly so striking and call for further study. The results of this form of prophylaxis in Gippsland are not available.

TABLE I.

Percentage Incidence of Visible Goitres in Canberra Children, Aged Nine to Eleven Years, at Each Examination.

Year.	Boys.		Girls.	
	Number Examined.	Percentage with Visible Goitre.	Number Examined.	Percentage with Visible Goitre.
1947	198	5·6	160	10·0
1948	236	2·1	215	5·0
1951[1]	140	nil	124	nil
1952[2]	299	nil	281	1·7

[1] Results from one school not available.

[2] Survey limited to children who had resided in Canberra for previous three years.

It is important to realize that iodine prophylaxis has no pronounced effect upon an already developed goitre. When a group of Canberra children with visible enlargement of the thyroid was followed for a number of years, it was found that although they regularly took 10 milligramme tablets of potassium iodide, there was little or no reduction in the size of the gland in the majority. However, the iodide did prevent the gland from becoming larger.

The regular consumption of tablets containing 10 milligrammes of potassium iodide in these studies has clearly demonstrated the effectiveness of iodine prophylaxis. However, the use of tablets has a number of disadvantages. The regular distribution and intake depend on the interest and application of a large number of people, some of whom display some indifference to the scheme. It requires constant and intensive supervision by people intensely interested in the outcome of the project. It caters for those who attend school regularly on the day of the week on which the tablets are given out by the teacher or monitor; thus it often misses the children most needing additional iodine—those from homes with poor family management.

For infants and pre-school children and pregnant women to receive additional iodine means regular attendance at a centre, and then someone in the household has to remember to give the child a tablet regularly. It does not provide for the adolescent who has left school, a group whose requirement is probably high. For these reasons there is much to be said for the replacement, in those parts of Australia where they are in use, of the provision of tablets of potassium iodide by a scheme which ensures that all members of the population will receive extra iodine as part of their normal diet. The use of tablets or any other individual medicament is impossible in New Guinea or any other undeveloped area.

The Safety of Iodine Prophylaxis.

On the subject of the safety of iodine prophylaxis, the meeting concluded that "informed opinion in all countries where iodized salt is used on a community scale is unanimous that no harmful results accrue and that the fear of iodine-induced hyperthyroidism is groundless".

The Choice of Iodine Compounds for the Iodization of Salt.

Up to the present potassium iodide has been the iodine compound of choice for the iodization of salt, candy or tablets, mainly because it is a common therapeutic agent. However, it is a relatively unstable substance which requires the addition of a stabilizer to the salt. Further, the stability of the iodine and of the stabilizer in salt is affected by the moisture, the heat and the degree of acidity of the salt. Moisture is probably the most important variable, for it causes the iodine to migrate through the container. If the moisture content is high and the package has stood for some time, all or almost all the iodide may be found in the walls of the container. If highly refined table salt is packed dry and kept dry in an impervious container, the iodide content will remain stable and relatively evenly distributed throughout the salt.

These conditions cannot be met except for a small fraction of the salt used in human consumption. The great majority of the world's population uses crude salt, some of it extremely crude, produced by solar evaporation of sea water or from inland salt pans. In endemic goitre areas where this form of salt is used, it has been necessary to find another way of iodizing salt. Under stimulus from WHO this problem has been studied in a number of ways during the past few years in several laboratories.

Iodates were selected for trial. They possess certain advantages over iodides, the principal one being their stability under adverse conditions of heat, moisture, sunlight and impurities in the salt. However, considerable concern was expressed in England by the Medical Research Council and in the United States of America by the Foods and Drugs Administration over the projected use of iodates, because earlier pharmacological studies had suggested that they were toxic.

Professor Margaret Murray studied this aspect of the problem and reported the results of her investigations to the meeting in London. Reexamination of the earlier experiments showed that the method of administration had been by injection, and the doses had been of the order of 500 times those likely to be given orally in the prophylaxis of goitre. Dr. Murray also reported briefly on certain studies made by Dr. Perry at the National Institute of Medical Research. He found that sodium iodate had a very low toxicity for mice and rabbits when given by mouth in doses comparable with those likely to be used in goitre prophylaxis. No pathological changes were found in either the kidneys or the retinæ, the organs affected in the earlier experiments. These studies suggested that iodization of salt at the rate of one in 1000 would be perfectly safe for the prevention of goitre.

Dr. Pochin reported to the meeting his studies on the availability of iodine as iodate to the thyroid gland. He found that almost all the iodine of the iodate was available to the gland.

The other question—namely, the effectiveness of iodates for the prevention of goitre—has also been studied in a well-controlled field experiment in Guatemala, in which tablets containing potassium iodate were used along with tablets of potassium iodide for the control groups. Iodates appeared to be as effective as iodides, and the London meeting recommended that iodates could be used for the iodization of salt.

Parallel with the clearance of iodates it was necessary to develop a technique for the iodization of crude salt. It was obvious that the technique of spraying iodides on refined salt on a conveyor belt would not be suitable for primitive salt plants in the economically underdeveloped countries. The Iodine Educational Bureau undertook to solve this problem, and they have developed a simple and easily operated process suitable for any area in the world where free-running table salt is unavailable.

The details of the apparatus, supplied by the bureau, are as follows:

> Coarse crystalline salt is fed into the hopper of a bulk controller at the rate of approximately five hundredweight per hour and passes into an enclosed wormscrew mixing conveyor which is 15 feet in length. At a point

approximately two feet from the base of the bulk controller a mixture of one part of powdered potassium iodate and ten parts of calcium carbonate or any available inert free flowing powder is introduced into the mixing conveyor through a precision feeder at the necessary rate to provide a final product containing the desired quantity of iodine. The salt and iodate mixture are intimately combined during their passage along the remaining 12-13 feet of the mixing conveyor and analyses have shown that the final product is satisfactory in every respect.

The wormscrew can be driven by any power unit—for example, electric motor or steam engine—or even manually.

Plans are available for the production of from five to 25 tons per day.

Need for Further Investigations.

The meeting expressed the opinion that every public health authority should accept the responsibility for determining whether endemic goitre exists in the population whose health it supervises. Endemic goitre areas should be carefully mapped. While moderate increase of thyroid size may occur without visible enlargement of the gland, for the purposes of survey work and for the assessment of the intensity of endemic goitre in an area, only visible enlargement should be recorded.

In the making of surveys the most satisfactory groups to study are children between the ages of six and fourteen years. Repeated surveys have found that the highest incidence in girls occurs in the twelve to fourteen years age groups and in boys in the nine to eleven years age groups.

In areas where iodine prophylaxis does not appear to be effective, much valuable information on the physiology of the thyroid in the people could be obtained with the use of isotopic iodide. The same techniques could be used to investigate further the suggestions made in earlier literature that a relationship exists between hardness of water, calcium intake and endemic goitre.

Summary.

The discussions of the meeting convened in London in December, 1952, by WHO to consider various aspects of endemic goitre can be summarized as follows:

1. Where the endemic goitre exists, food salt should be iodized at a level which will provide approximately 100 microgrammes of iodine daily to the consumer.

2. Where it is economically unpractical to supply the entire population with iodized salt, careful surveys should indicate the areas involved, and the salt should be provided to these groups.

3. Iodization should be legally mandatory.

4. Iodate can be used instead of iodide and should be the form chosen for the iodization of crude salt.

5. Many aspects of the problem still require to be studied.

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DIAGNOSIS BY THE SENSE OF SMELL.

By Charles Engel,
Sydney.

At the clinic of Budapest, where I worked as a young doctor, the patients were nursed by nuns. One of the nuns, Sister Alfonza, an elderly, kind woman with great experience, stands out most vividly in my memory because of one very remarkable and peculiar ability of hers: she smelt with great certainty the approach of death and very seldom failed to recognize typhoid fever by smell. When death was not instantaneous or dying was not too short, she was hardly ever wrong in the diagnosis. And concerning typhoid fever I make haste to state that it was not by the fœtor of fæces that she recognized the disease. I would not say that she possessed an abnormally keen scent; it seemed rather that by training and practice she had become adjusted and focused just to these two odours.

Moncrieff (1949) writes that "with practice a stage of development is reached, which is miraculous". I am thankful to the Sister as she aroused my interest in this kind of diagnosis, an interest which I have kept alive through the years.

In the literature of that time, the first decade of the century, I found little about the *"odor mortis"* (*"fœtor mortis"*, *"odor agonalis"*, "death smell"). Some information has been obtained from much earlier publications (Queckett, 1845; Isham, 1875). The odour has been described as not strong, but offensive, similar to that of the post-mortem room or the corpse. It is Rüdel's (1919) belief that this odour is the reason why the dog often turns away from its dying master.

Since that time I have come across a lot of cases in which the smell, some special characteristic odour, proved of assistance to me in early diagnosis, sometimes in a very decisive way. Limitation of space forbids me to describe them. So it appears to be beyond doubt that the sense of smell may be useful in diagnosis. Hippocrates knew that it was so, as it is clearly presented in his words: "The nose may also give good guidance in the diagnostics of the fevers, very often and in a spectacular way the odours being so different" (quoted by Beck, 1907). And over two thousand years later we read "a disease's odour might have been the major and final discipline in diagnosis" (Maclord and Witheridge, 1949). A great deal was written about it, especially in the last century. Later on, however, the pendulum swung in the other direction. Odours as diagnostic factors became neglected to a very considerable degree; I would even say, almost completely. Doctors lost interest in odours; they did not train the sense of smell at all—they rather disliked the idea. And in books odours were scarcely mentioned.

The explanation can be given very easily. The continuous unfolding and growing of medical knowledge and especially the substantial evidence given by the modern laboratories made earlier, better and more certain diagnosis feasible. I think that is the main reason, but there might be another reason too. People vary a good deal in their sense of smell (in two out of three it is fairly good) and react in different ways to the same substance. On the other hand, odours are very hard to describe, as we shall see later on. It is therefore inevitable that various authors describe the same odour in quite different ways. The reader of the description, the doctor himself, smells an odour which to his mind is quite unlike the description given in his books; so he becomes doubtful and sceptical and eventually forgets completely about odours in his practice.

I myself have long regretted the neglect of odours in diagnosis. I must say that I was quite amazed at the doctor who sniffed eagerly around the mouth of the patient with hepatic failure shown at a meeting last year.

But recently I have gained the impression that interest in odours is being awakened again. Within a decade two very important monographs (not strictly medical) have

been published about odours—the book of Moncrieff[1] (1949), already in the second edition, and that of Maclord and Witheridge (1949). The exhaustive bibliography compiled by Airkem Inc. came out about the same time (1952). And so I believe that osmics or osphresiology does not belong to the past as Boucher wrote in his paper in 1947.

With the present paper I hope to help to keep alive this newly aroused interest and possibly even to stimulate it. I think it is worth while. I would challenge the view that odours are only of academic interest in clinical medicine; I believe they have a great practical value as well. And I am firmly convinced also that to deal with odours is not an anachronism; I feel sure that they offer more than the general consensus of opinion would lead us to expect.

I shall first discuss the most important items of the physiology of smell and then continue with the odours under physiological and afterwards under pathological conditions in due order.

The Physiology of Smell.

The sense of smell is incomparably more acute than that of taste. There are four kinds of taste, but several thousands of odours. Several attempts have been made to classify the odours, but none of the systems obtained general approval. "Odours do not at this time lend themselves to precise classification."

It is generally very difficult to define odours. In words the description cannot be given well, or at all. The best way to characterize an odour is to liken it to some well-known fragrance or fœtor. So we hear about odours of violets, of bouillon, of rancid fat, of garlic, of mice, of decayed flesh *et cetera*. "Several odoriferous substances can combine to a new kind of odour, where the single components cannot be identified." Two or more odoriferous substances may cancel each other out. The sense of smell is rapidly fatigued for one odour, but can remain acute for others.

There are pleasant odours or fragrances and disagreeable offensive odours or fœtors and stinks. The English language is very rich in expressions for both (Devlin, 1946).

The odoriferous substances may come from the class of elements (iodine, bromine, phosphorus *et cetera*) and may also come from every class of compound. "There is no relation between the odour and the chemical formula of a substance." Very different substances may have similar odours, and quite a little change in the formula produces quite a different odour. "Even stereoisomeric substances may have quite a different quality of odours." The intensity of the odour decreases generally with the dilution, exceptionally the reverse may be stated (compare sulphuretted hydrogen). More important, however, is the fact that the quality of odour may change with the dilution. Many substances are pungent when concentrated and have just a bad odour when diluted. But even more important and surprising is the fact that a substance may have a bad smell when concentrated and offer a delightful scent when diluted. That is one of the reasons why the same substance may evoke different responses in different people with different nasal acuity. "The one with a poor sense of odour may smell the scent of big dilution whereas the man with keen scent feels the fœtor of the concentrated substance." The prototype of such a substance and the most important in this group is indole. Indole has a fæcal odour when concentrated, but in high dilution it is fragrant; the pleasant scent of some flowers (jasmine, wallflower, neroli) is due to traces of indole. "Triethylphosphine is suffocating when concentrated, when diluted it has the fragrance of hyacinth."

In the plant kingdom we meet mostly with pleasant odours produced by the plant cells. In the animal kingdom unpleasant odours are dominant, but we find pleasant ones too. They may be normal metabolic products (smell of urine, of milk, of seminal fluid) or abnormal ones (smell of acetone, *fœtor hepaticus*). But they are mostly produced by disintegration of organic substances mediated by viruses and bacteria in the tissues, bowels, cavities, abscesses, ulcerated surfaces of the mucosa and skin.

Different microorganisms produce different kinds of odours. In text-books of bacteriology we find many of the bacteria with the epithet "odoricus". Well known are the different odours of various kinds of cheese produced by different fungi. From among the common infective agents streptococci cause hardly any smell (not even in the lochia). More offensive may be the odour caused by some kinds of staphylococci, and even more so that caused by *Bacterium coli*. One of the most ill-famed stenches (ill-famed beyond the fœtor because of the high mortality of the disease too) is that produced by *Clostridium welchii*, which causes gas gangrene, to me the most terrible stench I have ever smelt.

In a large number of cases the chemical constitution of the odoriferous substance is well known; in others it has not yet been identified.

Odours in Normal Conditions.

As the dog recognizes its master by smell, every person must have his individual odour. The odour of every *secretum* and *excretum* may contribute to the integration of the individual smell. From this standpoint the smell of sweat is probably the most significant.

It has been taught that sweat has its offensive smell from volatile fatty acids, mostly from butyric, isobutyric and caprylic acids. In the text-books of chemistry, the odour of butyric acid is described as follows: "pungent in concentrated state but when diluted smells of perspiration. Isobutyric acid has even a more offensive smell" (Karrer, 1938).

The most recent studies of Barail (1947) "resulted in the most surprising discovery that in normal persons the fresh sweat has practically no odour at all. It is easily decomposed by bacteria on the skin and so acquires the characteristic fœtor." The more unclean a person is, the more bacteria are on his skin, the more repulsive his sweat becomes. As most of the sweat glands are in the armpits and in the anal and genital region, the sweat of these parts is the most unpleasant. To the malodour of the sweat considerable contribution is made by the decomposed *secretum* of the sebaceous glands (sebum) and to the sweat of the genital regions by the addition of secreta of the *glandulæ odoriferæ* (special sebaceous glands in this region).

The decomposition of these results in a very repulsive stink. Notorious and ill-famed is the stink of sweating feet. Fœtors originating from the shoes and especially decomposition of debris between the toes are contributing factors to the foul stink of sweating feet.

The odour of fresh urine is not unpleasant; it is aromatic, it smells like fresh bouillon. According to Dehn and Hartman (1914) the odoriferous nature of urine was erroneously attributed to volatile fatty acids. They found that one of the main odoriferous substances contributing to the odour of urine is urinod. Urinod is an ill-smelling substance, which, however, is present in urine in conjugated form. In fermentation it becomes free and gives the stale urine the characteristic bad smell which is surely not caused merely by ammoniacal fermentation of the urea. It may be caused, of course, by decomposition of proteins too. Moncrieff believes that according to the most recent examinations the odour of the fresh urine is mainly attributable to two steroid ketones—androstan-3-one and androstadiene-3-one; both have a distinct urine odour.

The urine acquires a very characteristic odour after the consumption of some food or drug. After the eating of asparagus the urine has the characteristic smell of methylmercaptan. After the taking of turpentine oil the urine gives off the smell of violets. This was known in ancient Rome and for a very long time it looked just mysterious. But "it is not such a long jump chemically as it is

[1] Every reader is referred to this excellent book. In the present paper I have quoted a lot from it. When sentences in inverted commas are not indicated otherwise, they are from this book. I have to acknowledge my indebtedness to R. W. Moncrieff for kindly permitting me to quote freely from his book.

olfactorily" (from limonene to ionon). I shall just mention the fœtor of cat's urine as some substances and disease odours are likened to it (favus). Only the urine of the male cat exhibits the characteristic stink because of mixing with the *secretum* of the *glandulæ odoriferæ*.

The characteristic, not too offensive, odour of the human fæces is mainly attributable to indole and skatole (methyl indole). Both are produced in the large bowel by disintegration of proteins, more precisely by decarboxylation and desamination of tryptophan (indole-propionic acid). After rich feeding on meat the odour becomes stronger, on pure milk diet the fæces have hardly any odour. That is the reason why the stool of infants on a milk diet has no fæcal smell; just a slight sour odour may be smelt.

Gas expelled from the rectum, flatus, is under normal conditions practically odourless because it consists mostly of swallowed air. It contains nitrogen (about 60%), methane (30%), carbonic acid (10%), some oxygen, hydrogen and just the slightest traces of volatile fatty acids, of indole and skatole. In cases of diarrhœa with abnormal fermentation and putrefaction the quantity of gas increases to a large extent and the gas has the offensive sour or foul fœtor.

Seminal fluid has a very characteristic odour. It is the *secretum* of the prostate that gives its smell. I have not come across any report of an investigation into the chemical constitution of its odoriferous substance. This has surprised me the more, as one can read that under special pathological conditions the fæces may acquire a distinct seminal smell (bacillary dysentery). Even the culture of dysentery bacilli (Shiga) may exhibit the smell.

The *secretum* of the preputial glands, smegma, has a very peculiar strong smell.

Tears, saliva, pure gastric juice and cerebro-spinal fluid are normally odourless.

Odours under Pathological Conditions.

In this section I shall dwell mainly on odours in diseases of my own specialty, internal medicine, and shall devote only some words to others.

The odour of the sweat of some persons may become abnormally offensive (osmidrosis, bromidrosis).

It is well known that difference in feeding may produce difference in odour of sweat. This may be the main reason why different races of mankind have different odours. In cases of bromidrosis the influence of feeding may be even more relevant. In exceptional cases of bromidrosis the fresh sweat may have this peculiar fœtor; mostly the fœtor develops as in normal persons, by decomposition of the sweat on the skin. Every person with bromidrosis has a special kind of malodour. The chemical constitution of the malodorous substance is not known. Sometimes drugs contribute to produce some characteristic bad smell (iodine, turpentine, arsenic *et cetera*). Bromidrosis in such patients is mostly associated with hyperidrosis, but may occur independently.

In urine on long standing the urea is split under the influence of urease from *Micrococcus ureæ* into ammonia and carbonic acid: $CO(NH_2)_2 + H_2O = CO_2 + 2NH_3$. Such urine has a strong ammoniacal odour. It is always an evidence of severe cystitis, if the freshly voided urine has the smell of ammonia. In constant trickling of the urine in man with retention of the urine, in woman with vesico-vaginal fistula the clothes have the characteristic urinous smell. It has already been mentioned that sometimes the very foul odour of urine is partly attributable to liberated urinod, but it may be caused by putrefaction of pus in the urine or by an ulcerated cancer of the bladder. In bacteriuria caused by *Bacterium coli* fresh urine may exhibit a very characteristic fœtor (fishy smell). In recto-vaginal fistula both the vaginal discharge and the urine may have a distinctly fæcal smell. One of the most loathsome and repulsive stinks in human pathology is exhibited by the pitiable women in whom ulcerated cancer of the vaginal wall has perforated into both the bladder and the rectum.

The normal gastric content, as already mentioned, has hardly any odour, it may just smell a little sour. In acute gastritis the vomitus may have a rancid or foul smell, especially if the gastric upset is caused by undigested and putrified food. The same rancid or rancid and foul odour may be exhibited by the copious gastric content vomited by patients suffering from a chronic pyloric obstruction with consequent gastric retention. In ileus, the gastric content may exhibit a just perceptible or a very pronounced fæcal smell. The contamination of the gastric content with fæcal substance can not only be smelt but may be detected on macroscopic examination too.

The odour of the fæces under pathological conditions is very important from both a diagnostic and a therapeutic standpoint. In diarrhœa the fæces may exhibit a rancid, sour fœtor (fermentation of carbohydrates and of cellulose) or a stenchy, foul one (putrefaction of proteins). The proteins which are putrified may come from the food, especially from meat, but may also originate from exudation of the inflamed intestinal wall, from massive bleeding into the bowel or from the surface of an ulcerated cancer. Sometimes the very offensive fœtor of the fæces cannot be properly accounted for when fermentation and putrefaction go on equally in the intestinal content. The odour of the fæces in itself may offer quick and very significant clues for treatment. In case of rancid smell the carbohydrates and cellulose, in foul smell the proteins have to be restricted or completely eliminated.

The fæces of cholera are practically odourless, those of bacillary dysentery may have (as already mentioned) the smell of seminal fluid or that of glue, but may have a very foul stench too.

If pancreatic juice does not enter the duodenum (chronic pancreatitis, obstruction of the pancreatic duct by stone or cancer), the fæces contain very big quantities of undigested fat which turns rancid in the colon. Copious, rancid-smelling fæces are a substantial evidence of chronic pancreatic disease. There are only two other diseases in which the fæces have about the same appearance and the same smell, sprue and cœliac disease. Because of lack of trypsin, decomposition of meat may also be present; in such cases the stools may have a putrid stink.

The bright yellow fæces of the patient with severe icterus are generally almost odourless. The fæces contain similarly much fat, but most of it is split, and it seems that in this condition it becomes rancid less easily than the unsplit fat.

Smell of the Exhaled Air.

On approaching the patient we easily detect the fruit-like odour of acetone in the breath of those with severe diabetes, especially in the precomatose stage and even more in coma itself. It may be smelt on entering the sick room. It may be of crucial importance in the differential diagnosis between diabetic coma and hypoglycæmic coma, in which such a smell is not present. It can also be noticed in patients with inanition without diabetes.

The breath of uræmic patients has sometimes a distinct urinous smell. According to Fishberg (1939) it is produced by the splitting of the urea secreted in the saliva. Dehn and Hartman believe that the splitting of the conjugated urinod may contribute to the *fœtor uræmicus*.

But the sweat of uræmic patients also contains urea, which may decompose on the skin and may increase the odour of the expired air.

Fœtor hepaticus is the name of the characteristic smell of the breath of patients with severe hepatic failure. It is described as sweetish, mousy, like new-mown hay, as the fœtor of decayed liver (Lichtman, 1949), of indole. It is probably caused by the autolytic disintegration of proteins in the necrotic foci of the liver, and so is produced by the same chemical process which causes the appearance of leucin and thyrosin in the urine of such patients. It cannot be smelt in every such case; Lichtman found it in twenty of a hundred cases of severe hepatic failure. It is of bad prognostic character, but it does not always mean a fatal outcome. E. Saint believes (personal communica-

tion) that it is almost as pathognomonic as the smell of acetone for severe diabetes.

The *fœtor mortis* already mentioned may be caused by similar disintegration of proteins. Usually such a disintegration takes places only after death. When the act of dying is prolonged it may, however, apparently precede death. Death is said to occur within ten hours of the appearance of the smell. As in the case of *fœtor hepaticus* only the positive finding is of diagnostic importance, though Rüdel thought that its absence meant that there was no imminent danger for the next twenty-four hours.

In practice the doctor very often meets patients with malodour of the mouth. The bad smell may be just perceptible or it may be very strong and offensive. So the fœtor may be just a slight nuisance, but it may become so repulsive that it makes the patients socially isolated. It can make their home, their office unlivable, in hospital it may require the strictest isolation. "The patient himself is hardly ever aware of the bad smell." Its cause can be sometimes very easily detected in some obvious disease of the mouth, the throat or the lungs. In other cases, however, its elucidation requires a good diagnostic acumen and the most thorough and meticulous examination. *Fœtor oris* can be caused by inflammation of the gingiva, *stomatitis ulcerosa, pyorrhœa alveolaris*, scurvy, inflammation caused by mercury poisoning, or *stomatitis gangrènosa*. It can be produced by ulceration of the tongue (cancer). It can be caused by diseases of the teeth—caries, gangrene of the pulp, decayed teeth with poor dental hygiene, or neglected denture. A very frequent cause is chronic tonsillitis. In this disease the little cheesy masses of debris, which can be easily expressed from the crypts of the tonsils, have the foul smell. The saliva can become fœtid by inflammation of the salivary glands. Ulceration in the throat may be a further cause (syphilitic, tuberculous or cancerous). Some authors describe the odour caused by diphtheria membranes as very characteristic (foul and sweetish). In their opinion it may decide the diagnosis. Other authors believe that it is not so specific as frequently stated (Nelson, 1951).

Malodour can be caused by diseases of the nose. It may come from manifest or latent retention of pus. Ill-famed is the very repulsive stench of patients with ozæna; it is surely one of the most unpleasant fœtors in human pathology. The French call the disease *"la punaisie"*; the fœtor reminds them of crushed bedbugs (*"la punaise"* in French). The foul smell is produced by putrefaction on the crusts in the sinuses. The purulent discharge from the ears in chronic otitis can become very offensive; in cases of cholesteatoma it may exhibit a distinct cheesy malodour.

Foul breath often accompanies disease of the bronchi and of the lungs. Well known is the malodour of the breath of patients with *bronchitis putrida*, bronchiectasis and especially gangrene of the lungs. It is important to know that the fœtor of the exhaled air may be much more intense than that of the sputum. I had often the impression myself that I smelt a not unpleasant odour on such patients, similar to that of flowers (jasmine, acacia). It is attributed to indole. F. Müller (1907) could identify it in some cases by chemical investigation. The breath becomes very offensive if a putrid empyema or a hepatic or subphrenic abscess perforates into the lungs.

I remember a couple of patients whose foul smelling breath was caused by stagnation and decomposition of food in diverticula of the œsophagus.

The breath can acquire a bad rancid or foul smell with acute gastritis. Similar fœtor may appear in the breath of patients with chronic anacid gastritis, but it is generally not strong in these cases. Much stronger may become the foul breath of patients with constriction of the pylorus and consequent retention of the stomach contents long before vomiting has started.

Massive bleeding in the bowels and ulcerating cancer of the colon may be the cause of foul breath.

Of crucial importance may be the foul smell of the breath of patients with ileus. I remember several cases in which it proved to be an early sign long before vomiting started. It is, of course, advisable in every case of suspected ileus to empty the stomach. The generally much stronger fœtor of the gastric content will well confirm the diagnosis.

Beyond all these it is quite sure and very important to know that simple constipation may produce a very foul breath.

Odours in Infectious Diseases.

The question of odours in patients with infectious diseases is not clear. It seems to me that there is no other field with such divergence of opinions. The odour in infectious diseases with ulceration and formation of pus and of membranes can easily be understood, as, for example, in diphtheria (already mentioned) or variola in the stage of pustulæ. Furthermore, the sickly nauseating fœtor of patients with yellow fever is quite understandable; it is somewhat near to the *fœtor hepaticus*. But unknown odoriferous substances produced by infectious agents in the course of abnormal metabolism of diseases must play an important role too. Such odours are described in simple infectious diseases even before the appearance of exanthemata and in infectious diseases without exanthemata. A number of authors report a characteristic smell in patients with measles, scarlet fever and typhus. The distinctly sour smell of patients with rheumatic fever is very often described. I mentioned in the introduction the certainty with which Sister Alfonza recognized the smell of typhoid fever. Alvarez wrote me in a letter than in olden times the doctors in America used to diagnose typhoid fever by smell.

I have already stated that I do not intend to deal in detail with odours in diseases of other specialties than internal medicine. I shall just mention that I have been taught by experienced gynæcologists (G. Santow) that they could smell the starting of menstruation well before the odour could have been produced by decomposition of the blood. Likewise they recognize the odour characteristic of vaginal discharge caused by fungi even before the patient undresses. What a bad smell of the lochia means to the obstetrician or the fæcal odour of the pus in the just opened abdomen to the surgeon is well known.

F. Goldschlag (personal communication) believes that beyond the already mentioned dermatological diseases the odour is of assistance to him in the differential diagnosis of the following diseases: favus (smell of mice or cellar); *condylomata accuminata* (reminiscent of gas in badly ventilated sewer); *pemphigus vulgaris* (honey-like smell); *pemphigus exfoliaceus* (decomposed moist foliage); *condylomata lata syphilitica* (offensive smell); plica (sweetish dull smell).

Finally I would mention how important the odour may be in differential diagnosis of poisoning, both in the living and at post-mortem examinations. In the new book of von Oettingen (1952) there are to be found very interesting detailed tables about this subject.

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THE INCIDENCE OF TUBERCULOSIS IN QUEENSLAND IN RELATION TO CLIMATE AND OCCUPATION.

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ONE of the factors postulated by Grotjahn in the early twentieth century as a basis for the application of social medicine was the following: "It must be determined how a disease of social importance, in the causation of which the social factor is involved, can be prevented by social measures or, alternatively, how it can be influenced in its course by them." However, before one considers preventing a disease, particularly in the case of a disease like tuberculosis, an accurate estimation must be made of the extent to which its frequency of occurrence and its course are influenced by different biological and social factors. In the following project an analysis of the incidence of tuberculosis in this State in relation to climate and occupation has been made, simple statistical procedures being employed to test the significance of the data obtained. I should like to point out that this project is not a detailed one, but is more in the nature of a pilot study, viewing the overall picture of quite an extensive subject. The data for this study were obtained as follows:

1. The cases of tuberculosis, with the patient's location and occupation, were obtained from the original notifications at the Department of Health, William Street, Brisbane, the four-year period, January, 1948, to December, 1951, being covered. Care was taken not to include in the survey more than once any cases which had been notified from two or more towns.

2. The mean temperature and the average annual rainfall for the towns used were obtained from the records of the climatological section of the Commonwealth Meteorological Bureau, Wickham Terrace, Brisbane.

3. Details of the populations in the towns estimated at June 30, 1951, were obtained from the Government Statistician's office, and the number of workers in each occupation came from the "Queensland Year Book" for 1950.

Tuberculosis and Climate.

In the grouping of the cases under headings of climate five main divisions have been chosen. They are as follows: (*a*) the tropical coastal strip north of Rockhampton; (*b*) the subtropical coastal strip south of Rockhampton; (*c*) the subcoastal area, which includes the highlands of the Great Dividing Range; (*d*) the far north-west, including Carpentaria and the Barkly Tableland; (*e*) the central and south-western parts of the State.

Two factors have been taken in the consideration of climatic conditions—firstly, the mean temperature, and secondly, the average annual rainfall. These two factors will be considered separately and then in combination. Towns in each of the five climatic divisions are used only if the mean temperature and the average annual rainfall are within fairly closely defined limits; for example, towns such as Cardwell and Tully have been excluded because of the extremely high rainfall. Rockhampton was not included because it is on the borderline of tropical and subtropical coastal divisions, and also because of the possibility of people taking up residence there owing to its proximity to Westwood Sanatorium. Other towns excluded were: Brisbane, because the metropolitan area has better facilities and many country people may come to Brisbane for confirmation of the diagnosis; Ipswich and a few other towns on the borderline between coastal and subcoastal areas; and Cooktown and Thursday Island, because of the high proportion of aborigines in the population.

On inspection of the figures in Table I there appears to be some difference between the number of notifications of the different climatic divisions, and to see if this difference is of any significance we apply to the figures the χ^2 test.

TABLE I.[1]

Town.	Average Annual Rainfall. (Inches.)	Number of Cases of Tuberculosis. (Total 264.)	Population. (Total 173,550.)
Tropical Coastal Strip.			
Mackay	66·91	46	14,600
Sarina	69·89	4	3580
Pioneer	71·51	2	12,740
Totals	—	52	30,920
Subtropical Coastal Strip.			
Bundaberg	43·47	8	17,200
Gympie	45·42	13	6880
Maryborough	45·63	30	16,310
Redlands	48·90	11	5840
Redcliffe	47·65	24	10,900
Pine	47·76	6	5070
Totals	—	92	64,200
Subcoastal Area.			
Warwick	27·49	9	8060
Emerald	24·61	6	2460
Charters Towers	24·64	17	7640
Dalby	26·12	5	5000
Kingaroy	28·82	7	8840
Duaringa	27·65	14	2040
Monto	29·37	10	4740
Stanthorpe	29·52	17	7780
Totals	—	85	46,560
Far North-West.			
Cloncurry	17·96	14	8400
Barkly	14·96	1	370
Hughenden	19·05	5	1950
Winton	15·90	2	2580
Totals	16·99	22	13,300
Central and South-Western Area.			
Charleville	19·38	4	12,070
Quilpie	11·11	1	
Cunnamulla	14·11	5	
Longreach	16·52	2	4310
Barcaldine	19·70	1	2190
Totals	16·16	13	18,570

[1] All the figures given, except the second set of occupation figures in Table XI, represent the total notifications for the four-year period January, 1948, to December, 1951.

From the data shown in Table I we set out Table II, of the observed frequency (*fo*) and of the expected frequency (*fe*), the latter being based on the null hypothesis—that is, the assumption that the results are of no significance.

$$\text{Thus } \chi^2 = \Sigma\frac{(fo-fe)^2}{fe} = 11{\cdot}9.$$

On taking this result to the χ^2 table we find that the differences between the observed and expected frequencies are significant between the 1% and 2% levels of confidence. As the results are highly significant, we now apply the figures to the rank difference correlation.

From this, ρ, the coefficient of correlation, is:

$$1 - \left(\frac{6\Sigma d^2}{n(n^2-1)}\right) = 1 - \left(\frac{6\times 10}{5\times 24}\right) = 1 - 0{\cdot}5 = 0{\cdot}5$$

Correlation is thus doubtful, since any coefficient greater than +0·5 or less than −0·5 indicates that a relationship exists between the two sets of data.

Now let us consider the incidence of tuberculosis and the effect of the mean temperature on it. In the preparation of this table rainfall has been disregarded, as is seen by the fact that a number of towns are included this time that were omitted in the rainfall table. Another feature is that for the subcoastal area we have limited ourselves to temperatures between 62·3° and 67·7° F., and this excludes Emerald and Charters Towers.

TABLE II.

	Tropical Coastal.	Sub-tropical Coastal.	Sub-coastal.	Far North-West.	Central and South-West.
fo	52	92	85	22	13
fe	47	98	71	20	28
$fo-fe$	5	−6	14	2	−15
$\frac{(fo-fe)^2}{fe}$	0·5	0·4	2·8	0·2	8·0

Again applying to the figures (Table IV) the χ^2 test and, as before, assuming the null hypothesis to be true, we have Table V.

Thus $\chi^2 = 50{\cdot}2$.

On referring this figure to the χ^2 table, we find that the result is significant at the 1% level of confidence, and so the differences are unlikely to be due to a chance distribution.

On applying the figures to the rank difference correlation we get the result set out in Table VI.

This time the coefficient of correlation, ρ, works out to 0·6, which signifies a fairly high correlation, and a relationship exists between the incidence of tuberculosis and the mean temperature of the environment.

We have tested as separate entities the significance of data in relation to average annual rainfall and mean temperature, so we shall now test their significance in combination. The towns chosen here are of necessity

TABLE III.

Rainfall. (Inches.)	Rank in Order of Rainfall.	Rank in Order of Cases per 1000 Population.	Rank Difference.	Difference.^{2}
16·16	5	5 (0·70)	—	—
16·99	4	3 (1·65)	1	1
20 to 30	3	1 (1·82)	2	4
40 to 50	2	4 (1·43)	2	4
60 to 70	1	2 (1·68)	1	1

fewer, as they will have to conform to two factors within reasonable limits. In order to have a better representation for the tropical coastal strip, Cairns has been included even though its average annual rainfall exceeds the range 60 to 70 inches. However, when the rainfall is around this figure small differences such as that in the case of Cairns are of little significance (Table VII).

If we assume the null hypothesis to be true, we have the figures shown in Table VIII.

Thus $\chi^2 = 59{\cdot}6$.

Taking this value to the table of χ^2 we once more find that the differences are significant at the 1% level of confidence. Rank difference correlation cannot be applied here unless one of the factors only is taken as our grouping; and as this has already been done for each of the factors separately, it will not be done again.

Tuberculosis and Occupation.

In considering the relationship between tuberculosis and occupation, we will consider only those subjects who are gainfully employed—in other words, those who are in receipt of wages. So we will exclude all cases in which the person is engaged in unpaid home duties, all pensioners (military, old age and invalid), all scholars and infants, and inmates of institutions.

TABLE IV.

Town.	Mean Temperature.	Number of Cases of Tuberculosis. (Total 460.)	Population. (Total 286,500.)
Tropical Coastal Strip.			
Cairns	76·5	60	18,500
Mackay	72·2	46	14,600
Johnstone	73·2	5	13,200
Townsville	76·0	87	36,600
Bowen	75·0	8	3540
Ayr	74·4	17	13,600
Totals	—	223	100,040
Subtropical Coastal Strip.			
Gladstone	71·5	15	6280
Bundaberg	70·1	8	17,200
Gympie	68·5	13	8880
Maryborough	69·8	30	16,310
Redlands	68·2	11	5840
Redcliffe	67·3	24	10,900
South Coast	68·1	26	16,600
Totals	—	127	82,010
Subcoastal Area.			
Warwick	63·4	9	8060
Dalby	66·3	5	5000
Kingaroy	63·7	7	8840
Gatton	67·7	5	6990
Cambooya	62·7	1	2090
Nanango	63·5	4	4500
Toowoomba	62·3	40	38,600
Totals	—	71	74,080
Far North-West.			
Cloncurry	78·0	14	8400
Barkly	76·9	1	370
Hughenden	75·1	5	1950
Winton	75·8	2	2580
Burketown	78·6	3	230
Totals	—	25	13,530
Central and South-Western Area.			
Charleville	69·9	4	12,070
Quilpie	70·9	1	
Cunnamulla	69·7	5	
Barcaldine	73·0	1	2190
Blackall	72·8	3	2580
Totals	—	14	16,840

The reason for this exclusion is that different factors may have entered into the causation of tuberculosis; in the case of housewives, previous occupations could have some bearing, and pensioners in many cases may be receiving the pension for tuberculosis. The grouping of the various occupations is taken from the "Queensland Year Book", as is the number of workers in each occupational group.

Assuming the null hypothesis we again apply the χ^2 test to the data from Table IX (see Table X).

Thus $\chi^2 = 61{\cdot}6$.

On taking this to the table of χ^2 we find that the observed differences are significant at the 1% level of confidence, so a relationship exists between the incidence of tuberculosis and occupation.

To test the reliability of the foregoing results I should like to include some figures obtained from the Chest Clinic, Department of Health, George Street, Brisbane. These figures (Table XI) concern tuberculosis patients discharged from public hospitals in Queensland during 1949, and were compiled at the Government Statistician's Office, Brisbane. As in the previous set of figures, housewives, pensioners, scholars and infants, and inmates of institutions will be excluded. To test the reliability of the data, a reliability coefficient of correlation between the two sets of figures will be determined by the use of rank difference correlation.

TABLE V.

	Tropical Coastal.	Sub-tropical Coastal.	Sub-coastal.	Far North-West.	Central and South-West.
fo	223	127	71	25	14
fe	161	132	119	21	27
$fo-fe$	62	−5	−48	4	−13
$\frac{(fo-fe)^2}{fe}$	23·9	0·2	19	0·8	6·3

Again, the differences are significant at the 1% level of confidence, as χ^2 was 81·3. However, we do not require this figure, since we are testing the reliability of the first set of figures. We now apply to both sets of figures the rank difference correlation to estimate the reliability coefficient of correlation of the two sets of figures, the ranking being according to case rate per 1000 employed in the group of occupations.

Reliability coefficient of correlation, ρ, is:

$$1 - \left(\frac{6\Sigma d^2}{n(n^2-1)}\right) = 1 - 0{\cdot}1 = 0{\cdot}9$$

As this value indicates high correlation, the two sets of data are closely related, and so the first set of figures is reliable and the differences in the different occupations are significant. As the highest case rate occurs in the manual

TABLE VI.

Temperature. (Degrees Fahrenheit.)	Rank in Order of Mean Temperature.	Rank in Order of Cases per 1000 Population.	Rank Difference.	Difference.[2]
62 to 67	5	4 (0·96)	1	1
67 to 70	4	3 (1·55)	1	1
70 to 73	3	5 (0·83)	2	4
73 to 76	2	1 (2·23)	1	1
76 to 79	1	2 (1·85)	1	1

group of workers, we will now analyse the figures for the separate occupations in this group to see if there are particular occupations which predispose the workers to tuberculosis (Table XIII).

As χ^2 is 125·4, the differences are significant at the 1% level of confidence, so within the manual group of workers there is a significantly high incidence of tuberculosis in a few occupations.

Discussion.

In considering the possible effect of climate and occupation upon the incidence of tuberculosis in Queensland, one must realize first of all that here we are discussing only those cases of tuberculosis which are notified, thus excluding cases which are not diagnosed because of the absence of signs and symptoms, or owing to inadequate facilities for diagnostic purposes, and others which for some reason are not notified at all.

Tuberculosis and Climate.

Firstly, we shall discuss the effects of the average annual rainfall upon the incidence of tuberculosis. The highest case rate per 1000 occurs in the subcoastal region, which has an annual rainfall of 20 to 30 inches. The next is the tropical coastal region, with a rainfall of 60 to 70 inches per year. Then comes the far north west region, followed by the subtropical coastal region, and last, the central and south-west region. All borderline towns were excluded, so a fairly representative group of towns was taken.

TABLE VII.

Town.	Average Rainfall. (Inches.)	Mean Temperature. (Degrees Fahrenheit.)	Cases of Tuberculosis. (Total 255.)	Population. (Total 152,260.)
Tropical Coastal Strip.				
Mackay	66·91	72·2	46	14,600
Sarina	69·89	74·8	4	3580
Cairns	88·75	76·5	60	18,500
Totals ..	—	—	110	36,680
Subtropical Coastal Strip.				
Bundaberg ..	43·47	70·1	8	17,200
Gympie	45·42	68·5	13	8880
Maryborough ..	45·63	69·8	30	16,310
Redlands	48·90	68·2	11	5840
Redcliffe	47·65	67·3	24	10,900
Totals ..	—	—	86	59,130
Subcoastal Area.				
Warwick	27·49	63·4	9	8060
Dalby	26·12	66·3	5	5000
Kingaroy	28·82	63·7	7	8840
Gatton	29·12	67·7	5	6090
Totals ..	—	—	26	28,890
Far North-West.				
Cloncurry ..	17·96	78·0	14	8400
Barkly	14·96	76·9	1	370
Hughenden ..	19·05	75·1	5	1950
Winton	15·90	75·8	2	2580
Totals ..	—	—	22	13,300
Central and South-Western Area.				
Charleville	19·38	69·9	4	12,070
Quilpie	11·11	70·9	1	
Cunnamulla ..	14·11	69·7	5	
Barcaldine ..	19·70	73·0	1	2190
Totals ..	—	—	11	14,260

Now let us look at the effect of mean temperature. The highest incidence is in the tropical coastal regions, with a case rate of 2·23 per 1000. Next comes the far north-western area, which incidentally has the highest mean temperature value. The others in order are the subtropical coastal area, the subcoastal area, and finally, the central and south-west area.

TABLE VIII.

	Tropical Coastal.	Sub-tropical Coastal.	Sub-coastal.	Far North-West.	Central and South-West.
fo	110	86	26	22	11
fe	61	99	48	22	25
$fo-fe$	49	−13	−22	0	−14
$\frac{(fo-fe)^2}{fe}$	40	1·7	10	0	7·9

It appears from these results that the combination of high average annual rainfall and mean temperature plays a significant part in the incidence of tuberculosis, the high mean temperature being the more important factor.

It is interesting to note that Thursday Island, which was not included in the foregoing series because of the large native population, has the highest case rate per 1000 in this State—namely, 13·84. This figure includes natives and European members of the population; but the high incidence is almost certainly due to the known susceptibility of coloured races to tuberculosis.

TABLE IX.

Occupation.[1]	Total Employed.	Cases of Tuberculosis.	Case Rate per 1000.
Rural Fishing Group: Hunting (0), Farming (65), Pastoral (12), Rural (8), Fishing (5)	109,075	90	0·83
Professional Group: Teaching (7), Medical (14), Religious (1), Scientific (1), Literary (1), Construction (1) ..	18,633	25	1·34
Clerical Group: Indoor Clerical (47), Administration (9), Other clerical (13), Selling (19), Agents (1)	87,307	89	1·02
Manual Group: Mining (9), Building (31), Metalwork (26), Leather and Stonework (7), Clothing (8), Food (6), Printing (6), Others (30), Labourers (77), Wharflabourers (21), Foremen (6)	140,115	227	1·62
Protective Group: Cooks (6), Stewards (1), Proprietors of cafés *et cetera* (6), Watchmen (4)	27,801	17	0·61
Totals	382,931	448	—

[1] Figures in parentheses are the number of cases of tuberculosis for the subdivisions of the occupational group.

A possible extraneous factor in the high incidence in the tropical coastal region is that the notifications may include patients from the southern States (New South Wales and Victoria) who have come to north Queensland during winter to avoid climatic conditions which may

TABLE X.

	Rural.	Professional.	Clerical.	Manual.	Protective *et cetera.*
fo	90	25	89	227	17
fe	128	22	102	164	32
$fo-fe$	−38	3	−13	63	−15
$\frac{(fo-fe)^2}{fe}$	11·3	0·4	1·6	41·3	7·0

superimpose other infection on their existing pulmonary conditions. However, these are very few according to the case register, and the numbers are too small to be of any significance.

It does appear probable, however, that tuberculosis may be more common in the hotter, wetter parts of the State than in the parts which have lower mean temperatures and lower average annual rainfall figures. Further investigation would be worth while when more data are available, if the foregoing factors could be eliminated as possibilities; and also if accurate diagnoses were assured by sputum examination and by X-ray examination of the chests of all subjects yielding a positive response to the Mantoux test.

Tuberculosis and Occupation.

In both sets of figures analysed tuberculosis is more prevalent among manual workers—skilled and unskilled, including all types of labourers—than in any other class of worker. Next comes the professional class, in which the incidence is undoubtedly higher because of the significantly high incidence in medical and health occupations, particularly among nurses. This is almost certainly due to the exposure of previously healthy girls to high concentrations of the causal organism in the tuberculosis wards of

TABLE XI.

Occupation.[1]	Total Employed.	Cases of Tuberculosis.	Case Rate per 1000.
Rural, Fishing Group: Farming (33), Pastoral (12), Timber (3)	109,075	48	0·44
Professional Group: Teaching (6), Medical (19), Religious (3), Literary (1)	18,633	29	1·56
Clerical Group: Proprietors (7), Manager (1), Officer (1), Clerical (47), Selling (19)	87,307	75	0·86
Manual Group: Foremen (4), Building (26), Metalwork (17), Engineers (7), Fitting (5), Others (53), Assistants (6), Drivers (7), Storemen (3), Labourers (46), Wharflabourers (8)	140,115	182	1·29
Protective Group: Police (1), Others (1)	27,801	2	0·07
Totals	382,931	336	—

[1] Figures in parentheses are the number of cases of tuberculosis for the subdivisions of the occupational groups.

the hospitals. This is a very important factor. The lowest case rate occurred in the protective and service group of occupations, and this is almost certainly due to the fact that here we are dealing with a selected group, since policemen, firemen *et cetera* have to pass a careful medical examination before admittance to the service. Another interesting point is the low incidence among rural workers, which would exclude contact with tuberculous cattle as an important factor in its cause.

TABLE XII.

Occupation Group.	Rank for Figures 1948 to 1951.	Rank for Figures Discharges 1949.	Difference.	Difference.[2]
Rural	4 (0·83/1000)	4 (0·44/1000)	—	—
Professional ..	2 (1·34/1000)	1 (1·56/1000)	1	1
Clerical	3 (1·02/1000)	3 (0·86/1000)	—	—
Manual	1 (1·62/1000)	2 (1·29/1000)	1	1
Protective ..	5 (0·61/1000)	5 (0·07/1000)	—	—

It is usually asserted in literature, particularly from Great Britain, that mining predisposes miners to tuberculosis. However, workers in this State hold that this does not occur in Queensland.

It is interesting to note that, whereas the over-all case rate for manual occupations is 1·63 per 1000, for miners it is 2·05 per 1000. The incidence is significantly high in labourers and wharf labourers, miscellaneous skilled workers and miners, and there appears to be some predisposing factor in its cause—for example, harmful dusts

TABLE XIII.

	Mining.	Building.	Metal.	Leather.	Food.	Wood.	Other.	Foremen.	Clothing.	Transport.	Labour.
fo	9	31	26	7	6	6	30	6	8	21	77
fe	7	34	48	5	22	16	7	5	17	13	53
$fo-fe$	2	−3	−22	2	−16	−10	23	1	−9	8	24
$\frac{(fo-fe)^2}{fe}$	0·6	0·2	10	0·8	11·6	6·3	75·3	0·2	4·5	5	10·9

in the case of miners, and exposure to chills and moisture in the case of labourers, who are often slovenly in their habits. This likewise would warrant further investigation, with a more detailed occupational history, and more accurate diagnosis in the doubtful cases, if the significance demonstrated from these data really exists.

Summary.

An analysis of the notification of tuberculosis cases from 1948 to 1951, inclusive, is carried out by the use of simple statistical procedures, and tests are made of the significance of the factors of climate and occupation in its occurrence.

1. There appears to be a prevalence of tuberculosis in the hotter, wetter climates—that is, in the tropical coastal strip. The area north of Rockhampton has the highest case rate per 1000 and the far north-western part is next. Some possible factors are discussed as predisposing causes, and a few possible complicating factors suggested.

2. Tuberculosis appears to be most common among manual workers, particularly labourers (including waterside workers) and miners. It is also of high incidence among the medical and health occupations in the professional class. Again, predisposing factors are suggested.

Both factors warrant further investigation with more accurate and detailed notifications from the sources of occurrence.

Acknowledgements.

I wish to thank Dr. E. W. Abrahams, Director of Tuberculosis in Queensland, for his interest in this project; Mr. J. Knight, research officer, Commonwealth Department of Health, who checked the results obtained; and Mr. E. P. Milliken, lecturer in vital statistics, University of Queensland, for his helpful suggestions and for making available some of the data required.

Reports of Cases.

CONGENITAL TOXOPLASMOSIS: A CASE REPORT.

By N. KERKENEZOV,
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THE first human case of toxoplasmosis was probably recorded by Janku (1923). Since that date fifty odd cases have been reported in widely scattered centres. To date I can find only six cases reported in Australia (Robertson, 1946; Hertzberg, 1952; Jack, 1952), and for this reason I think the following case warrants recording.

Clinical Record.

L.G., aged seven years, was first examined in September, 1952, as her mother was concerned about the child's vision. Both eyes had shown a rapid horizontal movement since birth, and the left eye was "turned in" from the age of six months. The child was a full-time baby and the delivery was normal; the birth date was August 11, 1945, and birth weight seven pounds 11 ounces. During infancy no abnormality was noted apart from the ocular signs mentioned earlier. The patient sat up and walked at the usual times, but never "talked properly". There was no history of convulsions.

When she was examined in September she had been attending school since 1949, but was unable to see the blackboard, and her progress was poor. Her general physical appearance and her activities were normal for her age. The visual acuity in each eye was "count fingers at three feet". There was a left convergent concomitant squint of some 15°, and she was unable to alternate. This condition was associated with a constant rapid, fine, horizontal nystagmus in all directions of gaze. Otherwise external examination gave normal results. The pupils dilated widely with atropine, and both lenses appeared normal. Fundal examination revealed a large patch of old chorio-retinitis involving both macular areas. The margins were sharply demarcated from the surrounding normal retina, and were fairly heavily pigmented. The lesions appeared punched out and characteristic of a coloboma (congenital) of the macula. Both disks were affected by a moderate degree of optic atrophy. A refractive error was noted, as follows: in the right eye, a sphere of +0·5 dioptre on a cylinder of −2·5 dioptres at an axis of 10°; in the left eye, a sphere of +2·0 dioptres on a cylinder of −0·5 dioptre at an axis of 10°.

An X-ray examination of her skull was reported on as follows:

> There are scattered areas of calcification in the left fore brain just anterior to the coronal suture line. The areas are about a cm. in length. No evidence of intracranial increased pressure is seen.

Serum from both the patient and the mother was sent to Dr. H. A. Feldman, of New York, whose report reads as follows:

> The serum of Mrs. G. contains antibodies for toxoplasma in a titre of 1:64 while her child is positive in a titre of 1:256. This should certainly support the diagnosis of congenital toxoplasmosis.

The mother was aged twenty-one years when the child was born. Prior to this she had had two children (now aged nine and eight years), and after this she had had a further three children (now aged six, five and three years). With all pregnancies she had had "kidney trouble all through" and "pleurisy at about six months". When she was pregnant with the patient under discussion there were a mother cat and three kittens in the house. I examined the eyes of all the other children mentioned above, but was unable to detect any external or fundal pathology.

The father, aged thirty-six years when the patient was born, is a vegetable grower, and had "boils and carbuncles" only during his wife's pregnancy with this child.

Comment.

In the case reported above, the association of ocular signs, cerebral calcification and a strongly positive titre for toxoplasma anibodies is, I think, ample justification for the diagnosis of toxoplasmosis. Of the three cardinal features—convulsions, cerebral calcifications and chorio-retinitis—only the first was absent. When the above-mentioned triad is encountered in infancy, the diagnosis is in favour of toxoplasmosis in the ratio of 9:1 (Sorsby, 1951). If the chorio-retinitis is absent in the presence of cerebral calcification and convulsions, then the diagnosis is in the ratio of 1:9 and most probably the condition is not congenital toxoplasmosis.

Acknowledgements.

I wish to offer my thanks to Dr. Harry A. Feldman (New York) for serum antibody tests, and to Dr. W. Pook and Dr. J. Byrne (Lismore) for the X-ray investigation of the patient in this case.

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Reviews.

Australia in the War of 1939-1945: Series One (Army); Volume I: To Benghazi. By Gavin Long; 1952. Canberra: Australian War Memorial. Sydney: Angus and Robertson, Limited. 9½" x 6¼", pp. 350, with about 84 illustrations and maps. Price: 25s.

IT will be remembered that the Australian official history of the war of 1939-1945 is being published under the title of "Australia in the War of 1939-1945". In this history there are five series, the first dealing with the Army, the second with the Navy, the third with the Air, the fourth with civil matters, the fifth with the medical aspects. The first volume of the medical series, entitled "Clinical Problems of War", by Dr. Allan S. Walker, was reviewed in this journal on December 13, 1952. The present volume is the first of the Army series, and the author is Gavin Long, the general editor of the whole of the history. We may state at once that this volume is produced in the excellent fashion which we saw in the first of Dr. Walker's volumes; it reflects great credit on the Advertiser Printing Office, Adelaide.

The first series dealing with the Army is to be published in seven volumes. Three of them will deal principally with the campaigns in the Middle East, and four with campaigns in East Asia and the Pacific. The author states that when he accepted the editorship of the history, it was decided that "the exercise of censorship by the government is to be limited to the prevention of disclosure of technical secrets of the three Services which it is necessary to preserve in the post-war period". The history is thus official in the sense that it has been financed by the Government, but that the writers have been given access to official papers and "have been conscious of the special responsibilities which rest upon writers of a national history". The volumes of the military series have been based chiefly on war diaries and reports of formations and units and some hundreds of interviews with participants obtained during the war, and on much correspondence and many interviews conducted after the war with a large number of soldiers. Mr. Long has had a difficult task. It is necessary that he should give a great deal of detail in regard to different units and their movements together with the names of their commanders and so on, and it will be readily understood that this tends to become wearisome to the general reader. However, the story in its general perspective is well worth while.

One of the most telling chapters in this volume is the first: "Between the Wars." Mr. Long shows how one government spent reasonable amounts of money on defence and another held different views. He paints a not too happy picture. At the end of his chapter he asks the rhetorical question what the Australian Government had done between the wars to carry out its military responsibilities. He points out in reply that in that period Australia had become a fully independent nation; she had greatly enlarged her colonial territory and had established her own diplomatic department. Her population had increased by nearly two millions and her industrial equipment had been vastly elaborated. There had been a corresponding increase in her responsibilities as a member of the British Commonwealth, and the "military leaders of the senior member of the Commonwealth" had declared that in a major war the immediate help of trained equipped forces from the Dominions would be needed. "Yet in 1939 the now-adult nation possessed an army little different from that of the young Australia of 1914." In an earlier part of this chapter Long states that it would be wrong to conclude that the Australian Army system had not achieved valuable results and that the devoted efforts of officers and men who had given years of spare time service had been wasted. "The militia had not and could not make efficient private soldiers, but it did produce both a nucleus of officers who were capable of successfully commanding platoons, companies and battalions in action, and a body of useful N.C.O.'s" The remaining part of this book shows how they did this and how the troops under their command took a nation's part in the conflict.

In spite of difficulties of various kinds, an expeditionary force was formed and the first convoy left Australia for Palestine, and not long afterwards other convoys followed. Australia, unfortunately, had two armies—the militia for service within the Commonwealth and the second Australian Imperial Force for service overseas. The Government had two objectives—to contribute forces to the British pool and to build up an army to meet possible attack by Japan. The Government therefore "found itself being pulled in two directions". Australia was unable to equip her armies from her own factories—there had been a failure to recognize that effective defence depended on a nation's ability to manufacture arms. One result was the appointment of a Director-General of Munitions Supply in the person of Mr. Essington Lewis, who was to act "with the greatest possible degree of freedom from ordinary rules and regulations". It was not long before Australia was able to supply a good deal of the equipment which she needed. When the first convoy sailed in January, 1940, Australia's military contribution to the Allied strength in Europe had been fixed at only one division. At the end of the year it had been expanded to a corps of four divisions. After the fall of France, it became evident that in the immediate future the military defence of the Middle East would have to depend increasingly on forces from India and the three Dominions east and south of Suez. Mr. Long gives many interesting details of the changes that took place. He describes the adamant attitude of General Freyberg of New Zealand and of General Blamey, the Australian commander, in their refusal to depart from the instructions of their respective governments. They would not consent to the piecemeal distribution of the units under their command, nor to their actual use until adequate training had been undergone by them. Of the Sixth Division Blamey said that it "must have training with the full scale (of transport) before it can be considered ready to take the field. I am very loth to allow this force out of my control until I am assured of its fitness for the task involved, and I am very certain that the Commonwealth Government will demand this of me". Another incident, not without significance to our story, must be told, as underlining "the individuality of the Dominion forces". General Wavell sent to Australian Corps Headquarters a copy of a proposed order to all troops under Wavell's command, in which it was stated that it had been decided to prohibit listening in to German broadcasts "in recreation rooms, etc.". Blamey passed the proposal on to his officers. Two of his brigadiers vehemently opposed the proposal. One of them thought that it would make the men suspicious of British broadcasts; the other seized on the opportunity to invite attention to the frequent obvious omission of unpalatable information from B.B.C. broadcasts. Blamey's reply to his superiors was that he thought the order would be unwise. He quoted as an example of the kind of omission that was likely to cause his troops to suspect their own news, the official report that one empty oil tank had been hit in the first Italian raid on Haifa, when the troops knew that several full tanks had been set on fire. Eventually, a somewhat modified order was issued, but Blamey was informed that the prohibition did not apply to the troops under his command. Long observes that any attempt to prevent Australians from listening to enemy broadcasts would not only have caused indignation, but would probably have been disobeyed by men who would have argued that they had enlisted voluntarily to fight for the preservation of just such a liberty.

Just before he begins to describe the actual fighting leading to Benghazi, Long ends a chapter with a paragraph which gives an indication of why Australian troops were successful in desert warfare.

> In the Australian force the special experience of its leaders as young infantry officers in the Australian Corps of 1918, or in the light horse in Palestine, had caused them to emphasise certain points in the British doctrines. The A.I.F. of 1918 had been rich in enterprising young officers promoted from the ranks; and throughout the training of the 6th Division emphasis had been placed on the need for encouraging initiative in N.C.O.'s and platoon leaders—a relatively easy task in an army in whose ranks were many potential officers. And, in 1918, the Australians had developed aggressive patrolling to a degree that had bewildered their enemy and astonished senior British commanders. It was a result of this earlier experience that the men of the new A.I.F. were given exacting training in patrolling, particularly at night, and were to go into battle convinced that, in all circumstances, they and not the enemy were to be masters of no-man's land.

The author takes us along the north of Africa and we can follow the battles for Sidi Barrani, Bardia, Tobruk, Derna and Benghazi. We shall make no attempt to trace the military progress in this area. The Italians had specially fortified Bardia and Tobruk and the history of the taking of these two places alone makes the book worth while. The terrain was difficult in some respects, but not in others. Describing the area near Bardia, Long writes:

> Few tracts of country anywhere presented fewer natural obstacles to the free movement of military vehicles; they could be manœuvred here as if in a great arena. On the other hand, the rough going caused rapid

wear and tear, and winds, drought, dust and extremes of temperature made campaigning uncomfortable even for troops well supplied with vehicles and gear. The wind might blow so strongly that it whipped up the harsh dust until for perhaps a whole day a vehicle five yards away would be invisible. In winter the nights were bitter, yet the days could be uncomfortably hot, and little shelter from cold, heat or wind was to be found. Yet to soldiers the desert offered two advantages: it was dry and free from disease; and the absence of towns and settled people over all the battlefield until Derna was reached meant that there were no refugees and no danger that civilians would be killed or wounded by bombing or gun-fire.

He writes elsewhere that the dust was sometimes dense—it was fine as talcum and would penetrate everything that was not tightly wrapped. The faces of men who had been working or sleeping in it would be powdered yellow-grey. It would sift into packs and food and choke the mechanisms of weapons. It could form so thick a fog that it sometimes brought traffic to a standstill.

The reader may be surprised to learn that Mr. Churchill and the Chiefs of Staff in London consistently regarded the advance in Libya as basically a defensive operation and thought that the purpose would have been achieved when "a safe flank for Egypt" had been secured, perhaps at Tobruk, perhaps at Benghazi. We read later that, in the light of present knowledge, if an advance had been made beyond Benghazi, Hitler would probably have ceased reinforcing the Italians, and the whole of Tripolitania could have been occupied. If certain judgements were correct, "a glittering prize was thrown away": the "possession of all Libya, air and naval bases opposite the narrow pass on the Mediterranean, the end of all danger of an advance on Egypt from the west supported by German arms, the isolation of French North Africa; perhaps the revival of French resistance". Churchill's eyes, however, were fixed on the project to form a front in the Balkans, and, in the event, his instructions were carried out swiftly in such a way as to put an end to all possibility of the resumption of the African advance that winter.

Long ends this volume with the statement that success gave the men increased confidence in their officers, in themselves and in their training; from among the younger men were emerging outstanding leaders "whom we will meet again in later battles". If this volume is read widely, as it deserves to be, those coming later will be awaited with interest.

Medical Mycology: An Introduction to its Problems. By G. C. Ainsworth, B.Sc., Ph.D., F.L.S.; 1952. London: Isaac Pitman and Sons, Limited. 9" x 6", pp. 114, with eight plates and seven text figures. Price: 22s. 6d.

In his "Medical Mycology" Mr. G. C. Ainsworth has managed to correlate a remarkable amount of information. Besides reviewing present knowledge in this little known field, he has, in each section, discussed the problems to be encountered, and the implications of host-parasitic relationships are clearly indicated.

As the author writes: "Both medical men and mycologists incline to the view that medical mycology is a confused and somewhat esoteric subject"; he obviously does not find it so, as his material has been presented clearly and logically. The introductory chapter gives a clear overall picture of pathogenic fungi, and is followed by chapters on diseases of the skin, the respiratory tract, the less superficial and the deep myocoses, serology, hay fever allergy and poisonous fungi. These last two chapters touch on aspects which are omitted from most American texts, but which should not be neglected. The book contains several excellent tables and a comprehensive bibliography, but could have supported more text figures. Interesting vital statistics are given.

Written from a mycological viewpoint, this book should help stimulate interest in this borderline branch of medicine. It is by no means a text-book, but is a very readable reference worthy of a place in any medical library.

Electrocardiography in Practice. By Ashton Graybiel, M.D., Paul D. White, M.D., Louise Wheeler, A.M., and Conger Williams, M.D.; Third Edition; 1952. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 11" x 8½", pp. 386, with 294 text figures.

Being familiar with the previous editions, we explore this book with confidence, knowing that it will be time well spent. It is compiled by authors who not only have a thorough knowledge of the science of electrocardiography, but also are familiar with its practical application.

Although it is founded on the former grounds, its special application is to the clinical side of cardiology. Paul White certainly is exceptionally well qualified to allot the relevant amount of attention to the various sections.

Being a practical book, it has a summary of the clinical details corresponding to each electrocardiogram, a feature which not only stresses the fact that the electrocardiogram must be considered as an integral part of the whole picture, but also stimulates the reader's interest and focuses his attention all the better on the records. An interesting feature is the concise historical record of the conception, birth and development of electrocardiography, which lays the foundation for the main part which follows. Experience has taught the authors the many variants of the normal and also the physiological changes which can occur. These occupy a considerable space, but familiarity with them will save the reader many an error.

The general division into chapters calls for no special comment, but hypertrophy and coronary disease are very adequately illustrated and discussed. The effects of drugs, electrolytes *et cetera* receive attention, but are not unduly stressed. In many cases serial tracings show the importance of the march of events. A final and very useful chapter is devoted to a series of unknowns on which the reader may test and consolidate his knowledge.

This book will prove a valuable friend to every practising cardiologist.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Diseases of the Throat, Nose and Ear", edited by F. W. Watkyn-Thomas; 1953. London: H. K. Lewis and Company, Limited. 10" x 7", pp. 892, with 367 illustrations, 22 plates in colour. Price: £5 10s.

The object of the book is to present a summary of the present views on the subject based as much as possible on individual experiences.

"History of the Second World War: United Kingdom Medical Series", Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S. "The Army Medical Services Administration", Volume I, by F. A. E. Crew, F.R.S.; 1953. London: Her Majesty's Stationery Office. 9½" x 6½", pp. 552, with 15 illustrations and six text figures. Price: £2 10s.

The first of two volumes in the "History" dealing with army administration; four volumes will be devoted to the army in campaigns.

"Modern Medical Monographs: Gout and Gouty Arthritis", by John H. Talbott, M.D.; 1953. New York: Grune and Stratton. 9" x 6", pp. 102, with 32 illustrations. Price: $4.00.

An attempt "to document significant advances in the understanding of the gouty discrasia and gouty arthritis".

"A Report on the Treatment and Care of the Elderly Chronic Sick in Bristol", by the Bristol Local Medical Committee; 1953. Bristol: Bristol Local Medical Committee. 10" x 7½", pp. 40. Price: 2s. 6d.

A report with summary and ten appendices.

"The 1952 Year Book of Neurology, Psychiatry and Neurosurgery (November, 1951-October, 1952)." Neurology, edited by Roland P. Mackay, M.D.; Psychiatry, edited by Nolan D. C. Lewis, M.D.; Neurosurgery, edited by Percival Bailey, M.D.; 1953. Chicago: The Year Book Publishers, Incorporated. 8" x 5½", pp. 604, with 121 illustrations. Price: $6.00.

One of the well-known Practical Medicine Series of Year Books.

"An Atlas of Surgical Exposures of the Extremities", by Sam W. Banks, M.D., and Harold Laufman, M.D., Ph.D.; 1953. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 11" x 8", pp. 408, with 552 illustrations. Price: £7 2s. 6d.

The authors aim at practical and anatomically correct surgical approaches, with emphasis on the preservation of function.

The Medical Journal of Australia

SATURDAY, AUGUST 15, 1953.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE WORLD HEALTH ORGANIZATION.

ON August 2, 1952, the work of the World Health Organization for 1951 was reviewed in these columns; the discussion centred around the annual report of the Director-General to the World Health Assembly and to the United Nations. On that occasion reference was made also to a discussion published two weeks earlier on an important book by N. M. Goodman dealing with the history of international organizations and their work. At first sight it may seem unnecessary to draw attention to the World Health Organization again after so short a space of time. But at least two reasons can be given. The first is that the health of nations is so inseparably bound up with their social and economic security that the latter cannot be attained without the former; the second is that many people in the community are not seized with the value and importance of these international organizations. We know beyond doubt that among the so-called leaders of our profession there are some who shrug the shoulder or turn a deaf ear when international medicine comes up for discussion; like the Pharisee, they "pass by on the other side". Let us then consider the annual report of the Director-General for 1952, published in March, 1953.[1]

This year's report gains added importance from the fact that it will be the last to be presented by Dr. Brock Chisholm, who retires from the office of Director-General in July, 1953. He was Executive Secretary of the Interim Commission of the World Health Organization from 1946 to 1948, and Director-General from 1948 to the present time. During his period of office WHO has made progress, and, reading between the lines, we feel justified in stating that a great deal of this is due to the energy and personality of the Director-General. On May 22, 1953, Dr. Chisholm delivered a farewell address to the Sixth World Health Assembly at Geneva, and reference will be made to this later. Turning first to the report, we note that in a foreword Dr. Chisholm states that the whole organization has now passed through its formative period, and that its general policies and procedures and its relationships, both internal and external, have been established. He also points out that a high standard of responsibility on a world level has been set up by the World Health Assembly and that relatively few intrusions are possible because of national or group interest or prestige. Ideally, of course, in such a body as this national or sectional interests should be able to obtain no advantage. The difficulty of achieving this state of affairs must be great, and the present confidence reposed in WHO is a reflection of its wise administration. Dr. Chisholm declares that perhaps the most important demand that WHO makes on its member governments is that they should ensure complete world-mindedness in the members whom they designate to the Executive Board and that then they should respect the absolute independence of those designated. This will be possible only if the peoples of the world and the governments representing them recognize that WHO has been formed so that the stronger nations may help the weaker to become healthy and efficient contributors to the well-being of the nations as a whole.

The present report, like last year's, is divided into three parts. The first deals with the direction and coordination of international health work, the second with the assistance given to governments in the regions, and the third with collaboration with other organizations. There is a useful introduction devoted to a discussion on trends in world health. The point is made here that health is part of economic and social development, a fact which is realized by most students of preventive medicine. The reader is reminded that it has been characteristic of the more prosperous and highly industrialized countries that improvements in health have gone hand in hand with social and economic advancement. As a matter of fact, as already implied, if improvements in health are not accompanied by corresponding advances in other fields, they can be little more than "ephemeral or piecemeal". In most of the less industrialized countries lack of capital constitutes a serious obstacle to any general advance. This means that these countries must have the means of developing their own resources; until this is possible, "any appreciable raising of the level of health . . . is financially impracticable". It is suggested that a partial solution may lie in the programme of "Technical Assistance for Economic Development" which "by strengthening national health services within the general context of economic advancement, could provide the means of realizing lasting benefits to health". But for this limited purpose the amount available is far from adequate. The Economic Commission for Europe studied this subject and calculated first that to meet the present rise in population in under-developed countries and secondly that to permit the modest increase of 2% annually in their *per capita* income would require a flow of sums well in excess of ten billion dollars a year from the developed to the under-developed countries. It is most satisfactory that, in spite of this almost unattainable state of perfection, as we may call it, so much has been

[1] "The Work of WHO, 1952: Annual Report of the Director-General to the World Health Assembly and to the United Nations." Official Records of the World Health Organization, No. 45; 1953. Geneva: World Health Organization; Australia: H. A. Goddard Pty., Ltd., 255A George Street, Sydney. 11" × 8½", pp. 214, with 17 plates and 11 maps. Price: 9s.

and is being done with the limited means available. On the important subject of communicable diseases it is stated that in well-developed countries the incidence of many diseases has been reduced. Virus diseases create a special problem, because in most cases there is no effective means of controlling them. WHO, in its attempts to reduce the toll of the major endemic and epidemic communicable diseases, has concentrated on improving resistance to them, controlling the vectors, reducing animal or human reservoirs of infection, and encouraging research directed at improved methods of control. It has also provided training for such work. Continued successes have been achieved in the mass control of the treponematoses by treatment with penicillin and in the control of malaria and other insect-borne diseases by the use of residual insecticides. Since the effective use of insecticides is limited, WHO has stimulated research into the reasons why certain vectors become resistant and others do not. Vector control, as distinct from insect control, has advanced in many parts of the world. WHO has also been active in the international coordination of research and in the standardization of laboratory procedures. The Tuberculosis Research Office has been active and a special monograph on B.C.G. vaccination in Copenhagen has been published by it. The activities of WHO in the strengthening of national health administrations are described. During 1952 it assisted 22 governments in the organization of public health services, by health surveys, the initiation of rural health centres, the planning and establishment of health demonstration areas, and general advice on national and local health services. Activities on nursing, on maternal and child health, on mental health, on social and occupational health, and on nutrition are among the other activities described.

It is impossible to give in this place an adequate idea of the work of WHO. Those who desire detailed information must seek it in the report itself. What is needed at present is that people, especially medical people, in the member nations shall know that, as Dr. Chisholm tells us in his valedictory address, WHO has been turned into a going concern; its concepts have become living reality. It has become "an organization which can see to it that the internationally available means, so badly needed for improvement in health and so limited in scope, are used in the most economical and effective manner". At the present, Chisholm states, WHO is spending on international action for the protection of the health of men, women and children of the world no more than the amount many a large city spends on its own municipal sanitary arrangements. We must agree that the amounts spent are ridiculously small when viewed against the background of the health needs of the world in which two-thirds of the population are still deprived of the benefits of modern scientific knowledge. Chisholm ends his address with an eloquent appeal in which he declares that man must learn to live with himself and get along with all others in a world in which dimensions and perspectives are radically changed—"he must reshape his entire pattern of thinking and behaviour in order to build a completely new system of human relationships adjusted to a changed world". Humanity is caught in a vicious circle. Dr. Brock Chisholm cannot resolve it. WHO cannot resolve it. No government can resolve it. The enigma remains one for individual men and women in different parts of the world who can think along constructive lines and induce others to do the same.

Current Comment.

PAIN AND SHOCK IN CORONARY OCCLUSION.

ALL is not said about pain as a clinical symptom of coronary occlusion when its nature and distribution are described, for other curious features have been observed. For example, it is not necessarily easy to explain that symptoms of shock are predominant in some patients and not in others. There are, of course, many factors related to individual vascular and nervous systems which may account for these differences. Since the time when the first accurate clinical and electrocardiographical studies were related to the morbid anatomy of the heart and vessels it has been occasionally noted that pain, persistent or readily evoked by different types of effort, may disappear after the subsequent occurrence of a proven myocardial infarction. This has been thought to be due to the death of an area in the heart tissue which previously was hypersensitive and prone to provoke an anginal reaction. John S. Gilson and Cecil M. Day have investigated several cases of this nature and have come to the conclusion that the initial pain, even though prolonged or persistent, was due to ischæmia, and that its disappearance was caused by the loss of all excitatory power by the previously sensitive cells.[1] They have put this to the proof by taking electrocardiograms during both episodes, and report that the tracings during the painful episode were those generally recognized as of ischæmic origin, whereas those recorded during or after the second occlusive attack were characteristic of myocardial infarction in one of the familiar patterns. One of their six investigated patients showed the ischæmic graphic pattern even at a time when no pain was felt, and another had the so-called "shoulder-hand syndrome" before the eventual infarction, but two months after the original occurrence of anginal symptoms. In all their recorded cases severe and readily evoked pain was fully relieved only when a proven infarction took place. The authors have been able to verify the findings by autopsy in one instance. It will probably be admitted that the cause of "coronary" pain is ischæmia, but some caution is advisable in the localization of the site of an ischæmic arterial lesion.

Another important clinical feature associated with myocardial infarction is the onset of severe peripheral shock. Clinicians have sometimes inquired by what mechanism this is produced, and have wondered if it was to some extent protective. Here we are perhaps apt to be confused by a mixture of physiological and philosophical reasoning, but we must have some scientific basis for treatment of this emergency. T. R. Fink, C. J. d'Angis and S. Biloon in an article on the subject describe their findings in a series of 15 patients suffering from severe peripheral shock following coronary occlusion.[2] They remark that shock in acute myocardial infarction carries a very bad prognosis, and consider therefore that the question of treatment is urgent. The majority of their shocked patients were found to have venous pressures above the normal average; only the minority had venous pressures below the normal. These latter were also pale, they sweated and appeared about to die, whereas the higher pressure group were less disturbed, though their skin was cyanosed. The authors, thinking of the analogy with traumatic shock, tried the effect of administering plasma intravenously, though they thought that the consequent raising of blood volume would be dangerous. They attempted to reduce the size of the vascular bed by giving "Neo-Synephrine" which they thought an appropriate drug, as it does not produce tachycardia or coronary constriction, and is brief in action. They used a digitalis preparation in the high-pressure

[1] *Ann. Int. Med.*, March, 1953.
[2] *J.A.M.A.*, April 4, 1953.

group, and found it raised arterial and lowered venous pressures. They thought the use of plasma was contra-indicated. We must admit that the validity of treatment is difficult to establish in so grave an emergency.

These two subjects are unrelated except by their occurrence in a common and serious condition, but they are alike in raising some physiological observations which should constantly be in the mind of the physician who is responsible for immediate treatment.

LIQUORICE AND CORTISONE.

ONE of the characteristic properties of some of the adrenal cortical hormones is their action on the kidney whereby sodium, chloride and water (extracellular fluid) are retained while excretion of potassium is increased. When administration of the hormone is stopped there is a rebound, potassium is retained and extracellular fluid is excreted in excess. It has been noted for some time that a large percentage of patients taking liquorice developed œdema. Molhuysen *et alii* in 1951 described retention of water, sodium and chloride and increased output of potassium after the oral intake of extract of liquorice or the glycyrrhizinic acid derived from it; in other words these substances had the same effect as deoxycortone.

Groen *et alii* in 1951 claimed to have controlled a case of Addison's disease with liquorice. J. G. G. Borst, S. P. ten Holt, L. A. deVries and J. A. Molhuysen have made a further examination of the effect of liquorice in Addison's disease.[1]

The deoxycortone-like effect of liquorice was found to be absent in three patients with Addison's disease. One of the three had suffered from suprarenal insufficiency for only a short time and had at first reacted well to liquorice. In these patients the administration of 2·5 milligrammes of cortisone every day partially restored the effect of liquorice on the mineral metabolism; 10 milligrammes of cortisone restored it fully. The effect of deoxycortone was not increased by cortisone. It is suggested that liquorice is active only in the presence of glycocorticoids and that patients with Addison's disease who respond favourably to liquorice still have remnants of functioning suprarenal tissue.

In the same number of *The Lancet* W. I. Card, W. Mitchell, J. A. Strong, N. R. W. Taylor, S. L. Tompsett and J. M. G. Wilson give the results of a similar study. They confirmed the effect of liquorice and glycyrrhetinic acid in normal subjects and studied one case of Addison's disease in which the patient was treated with glycyrrhetinic acid. In this patient the effects observed were similar to those seen with cortisone and deoxycortone therapy. On stopping the medication there was prompt reversal of these changes. That the patient was not wholly without functioning adrenal tissue is seen by the fact that she was discharged from hospital without maintenance treatment but returned to hospital later. Crude liquorice and glycyrrhetinic acid did not prolong the life of adrenalectomized rats while deoxycortone did. It seems from these studies that some constituent of crude liquorice has a synergistic action with certain of the adrenal cortical hormones.

MALIGNANT MELANOMA.

AT the beginning of a discussion in these columns on malignant melanoma of the skin in the issue of September 13, 1952, the subject was introduced with a quotation from the late E. H. Molesworth, describing melanoma as "one of the most viciously malignant tumours that affect the human body". These are words which should always be borne in mind whenever melanoma comes up for discussion. In last year's article we drew attention to a classification of pigmented moles by F. H. Weidman. He described seborrhœic keratoses, the blue nævus, the common brown mole and the blue-black pigmentary nævus. The last-mentioned was stated to be the one of dire significance. Weidman also mentioned the lentigines, a term connoting freckles. These were potentially malignant; they were the multiple moles which crop out in childhood or early adult life, are distributed widely and haphazardly on the body, and are variously brown to black in colour. In discussing prognosis, Weidman pointed out that the ways of malignant melanomata are as unpredictable in this as they are in many other respects.

With Weidman's contribution in mind, we would refer readers to a report recently published on the end results in the treatment of malignant melanoma. The report covers 1190 cases, and is made by G. Pack, D. M. Gerber and Isabel M. Scharnagel.[1] These cases of malignant melanoma were observed over a period from January, 1917, to the end of December, 1950. The authors claim that it is the largest series of malignant melanomata ever analysed and reported. The entire group of patients were under the management of one department with group judgement and control; the span of years during which diagnosis and treatment were carried out was long, affording an unusual opportunity to evaluate the results of therapy. These authors in their report use the term "melanoma" to signify the malignant tumour only. They do not include the compound or junctional nævus. Further, prepubertal or juvenile melanoma is not included in the discussion, as its clinical behaviour is usually benign. It is important to note that throughout their study histological confirmation of the clinical diagnosis of malignant melanoma was obtained in every instance. The essential points about this study will be stated at once. The reasons for the importance of melanomata are set out by the authors as follows: (i) The majority of these "black cancers" originate from long-standing preexistent nævi. (ii) The majority of melanomata could be prevented by the judicious excision of dangerous nævi. (iii) The constant visibility of the tumours should enable an early diagnosis to be made. (iv) Although the melanoma is the most accessible of all forms of cancer, it has the unwelcome distinction of having the lowest curability. The authors' report deals with 1190 cases, but the number of those finally dealt with is considerably less. In the first place, all patients seen within the last five-year period 1946 to 1950 had to be omitted; these comprised 446 new patients. This left 744 patients to be considered. These are divided into what the authors call a determinate and an indeterminate group. Only the determinate patients are considered; those branded indeterminate included patients who were lost to the follow-up within a five-year period of treatment, patients who refused treatment or palliative care, and patients who, within a five-year period after treatment, died of some cause other than malignant disease. Of the 575 patients available for analysis, 123 or 21·4% were free from evidence of melanoma five years or more after treatment. Some encouragement is rightly drawn from the fact that the end results show a steady improvement as the five-year periods are passed. Thus, with the 1917-1925 group the percentage of cures was 10·0; with the 1941-1945 group the percentage was 26·0. Some practitioners may object that a period of five years without recurrence is not necessarily evidence of cure of malignant disease. They will possibly call to mind cases in which the patient has lived for twenty years after, say, surgical removal of malignant disease of the breast, and has then suffered from a recurrence perhaps in the sternum. This view is undoubtedly correct, but at the same time our authors point out that in melanoma, probably more than in most other malignant tumours, the five-year post-operative period without evidence of recurrence *is valid as a criterion of cure. At the same time they* admit that there are a few unusual instances of patients who have survived for more than five years with residual melanoma present. They refer to one patient who lived for seventeen years after local excision of melanoma and repeated surgical removal of recurrences, and who finally died with widespread metastases. It was found that the results varied according to the sex of the patient. The

[1] *Lancet*, April 4, 1953.

[1] *Ann. Surg.*, December, 1952.

percentage of cures was 27·4 for women and 15·6 for men. Various reasons for this are discussed, but it is concluded, probably correctly, that women as a group are more "cancer-conscious" than men. Women tend to see a doctor earlier because of a mole which is changing in appearance. There was practically no difference in prognosis according to age after the onset of puberty.

In the matter of treatment some interesting facts are disclosed. Of the malignant melanomata, 73·3% fell into the operable group. It is stated that delay for more than a month in the institution of surgical operation after adequate local excision reduced the five-year cures from 39·9% to 17·0%. When metastases were present in regional lymph glands, the figure for five-year end results was 14·1%; when no lymph glands were affected, the percentage of five-year cures was 40·5. The authors rightly insist that the importance of obtaining proper surgical treatment of melanoma without delay cannot be over-emphasized. They refer to a contribution by two of them with another author on the principle of excision and dissection in continuity for primary and metastatic melanoma of the skin. This will be found in *Surgery*, Volume XVII, 1945, at page 849. We do not propose to do more than refer to this contribution; those who are interested will consult it for themselves. If this article by Pack and his co-workers does nothing else, it will emphasize the importance of early diagnosis and also the fact that when a melanoma is discovered a counsel of despair need not be adopted.

A NEW DRUG IN THE TREATMENT OF PERIPHERAL ARTERIAL VASCULAR DISEASE.

THERE is perhaps something of the therapeutic experiment in the use of some of the newer drugs in the treatment of peripheral vascular disease. This does not mean that the administration of such drugs has no defined scientific basis, but rather suggests that after extensive pharmacological investigation clinical use of the drugs is promising, and that they may be used by comparison with others of the same type. In particular such preparations, if effective, may help us to evaluate the possible justification of one of the procedures of vascular surgery. The use of the word "new" is attended by some degree of uncertainty, too, for it is only relative. In fact it is likely that there are yet promising pharmacological substances lying in idleness, though one hesitates to suggest that the zeal of the synthetic chemist may have outrun his discretion. Harold D. Green and William T. Grimsley have made a study of the effects of "Regitine", a new anti-adrenergic drug which was first developed in 1947.[1] It is of the same pharmacological group as "Priscoline" and "Etamon", whose trade names are more familiar.

Laboratory work has been carried out by several observers on this drug, and the present authors felt that clinical trials in man were fully warranted. They studied 34 patients who were divided into four groups; 10 were suffering from Raynaud's disease, 14 from peripheral arteriosclerotic vascular disease, all but one being males, three from *thromboangiitis obliterans*, and seven females from miscellaneous disorders unrelated to vascular disease. When a specific diagnosis was made it was based on careful observations and satisfactory definitions. The effect of "Regitine" was investigated first on the skin temperature of the subjects, because both vasospasm and organic occlusion are usually factors in producing the signs and symptoms of vascular diseases, and measurement of the skin temperature changes gave a standard by which the degree of antispastic action of a drug could be measured. These studies were made by the use of thermocouples, care being taken that the patients were in a room with constant air temperature, and that the subject's skin was protected from undue air flow or other disturbing conditions. Some studies were also carried out with "Etamon", to contrast the effects observed. "Regitine" was administered by the intravenous route in the skin tests, but for the therapeutic trials the oral route was used. The evaluation was to a good extent subjective, but objective response was looked for where possible in such phenomena as healing of lesions, better growth of the nails, and increased powers of walking. The results of the test showed that the drug has both adrenolytic and sympatholytic properties; it could overcome spasm due to cold as well as "Priscoline". Comparison of its effect on skin temperature with that of "Etamon" was useful chiefly as a standard of comparison whereby the value of the drug could be predicted. Oral administration of 30 to 120 milligrammes from once to four times daily was carried out for periods ranging from two weeks to fourteen months. The results were somewhat different from those which might have been expected in some regards. Thus patients with arteriosclerotic disease appeared to be benefited more than those with Raynaud's disease. Few side effects of any great magnitude were noted: vasomotor collapse was observed, however, in two instances. Over 60% of the subjects complained of minor manifestations, such as looseness of the bowels, palpitation, nausea, chilliness and other symptoms of that order. The patients with severe reactions soon recovered with appropriate treatment.

The authors conclude that the drug appears to be useful as an adjunct to other measures taken for vascular disease of the peripheral type, and think that it would be of value before a more drastic line of action such as sympathectomy was advised. They also remark, with a touch either of caution or of pessimism, that it is useful in the treatment of patients who have undergone sympathectomy. There is already a fair range of drugs of this type, and this may be added as another of those which appear to be reasonably active and reasonably safe.

[1] *Circulation*, April, 1953.

TREATMENT OF ACUTE RENAL FAILURE.

THE modern surgeon must know his physiology, or certain parts of it, if he is to be more than a specialized craftsman, and in no field is this more true than in that of urology. As S. A. Vest and R. A. Kelley[1] put it, the time has come for all urologists to make themselves proficient in all modern conceptions of renal physiology and in the problem of water, electrolyte and protein balance. In a paper on the treatment of acute renal failure they point out that uræmia has long been thought to be a syndrome resulting from retention of waste products of protein metabolism, but it is far more complex than failure to excrete any single nitrogenous substance. The most important disturbance in renal insufficiency is failure of the kidney to maintain a proper volume of body fluid and chemical equilibrium between various electrolytes within the plasma and other fluid compartments of the body. Disturbances in fluid and electrolyte balances account for most uræmic symptoms. Proper maintenance of fluid and electrolyte balance is therefore the cardinal therapeutic objective. In Vest and Kelley's paper the question of post-renal (obstructive) anuria is not considered. The problem studied is acute renal failure of the anuria or oliguria type, as a result of pre-renal or renal disorders. As regards fluid balance, it is stated that with the suspension of kidney function, water will be lost only through the so-called insensible channels. Under average daily conditions, there is an insensible loss of about 1000 mils. When the external temperature is high, or fever is present, the loss may be doubled or even tripled. Loss by diarrhœa or vomiting must be added. When the patient is first seen, a clinical estimate of his state of hydration should be made. Dehydration, even with normal kidneys, is inimical to the resumption of normal renal function and must be corrected. Over-hydration, however, is dangerous, resulting in pulmonary œdema and cardiac failure. As regards electrolyte balance, the plasma electrolyte concentration should be maintained as near to normal as possible for proper cell functioning and prolongation of life. The

[1] *J. Urology*, January, 1953.

administration of sodium in any form is rarely indicated, except when there is a deficit, as after vomiting or diarrhœa. The loss of sodium from normal perspiration or bowel movements is insignificant. Plasma sodium values should be frequently determined. A lowered value results in shift of water from the plasma into the cells, causing intracellular œdema. On the other hand, an elevated plasma sodium value, such as that following injection of saline, lactate or bicarbonate, causes a shift in the opposite direction, with consequent hydræmia and danger of pulmonary œdema and cardiac failure. With this goes an expansion of the extracellular compartment, and this further embarrasses the circulation. Another important consideration is the potassium factor. Sudden deaths in anuria are probably due to cardiac arrest from potassium poisoning. As 90% of potassium is excreted by the kidneys, failure of kidney function can lead to hyperpotassæmia; but it will not occur if at least 200 mils of urine are being excreted daily. The important substance for ingestion, or for intravenous therapy, in anuria is glucose, not so much to supply Calories as to reduce ketosis and protein catabolism. This causes diminution in the amount of potassium liberated by the cell. As it is theoretically ideal to keep the patient in caloric balance, fats can be added to the diet by giving a mixture of peanut oil and glucose to provide 2000 Calories per day; this, however, may produce severe diarrhœa, with water and electrolyte loss, and it is better for the patient to be in caloric deficit for two weeks than for this mixture to be forced too much. If the glucose cannot be tolerated when given orally, it is given in a dose of 50 to 100 grammes daily by the intravenous route. Proteins and fruit juices, most of which contain potassium, are to be avoided. Diuretics have no place in the plan of management, as they can cause disastrous disturbance of the plasma electrolyte pattern.

Vest and Kelley state that with the plan of management outlined, there is an onset of diuresis by the end of the second to third week of the anuria. Time has been allowed for renal repair. As regards accessory treatment, they consider that far too many optimistic claims have been made for internal or external dialysis, and that in any case, such dialysis is rarely necessary in the first two weeks of anuria. Technical difficulties are numerous with external "Cellophane" dialysis (artificial kidney) and also with peritoneal dialysis. Intestinal dialysis, especially with a Miller-Abbott tube in the jejunum, is efficient in removing potassium and urea from the plasma.

Finally, it is pointed out that when diuresis becomes established, a vigilant watch should be kept for the excessive loss of salt during this period and its replacement attended to, if necessary.

YAWS.

YAWS is still prevalent in various tropical and near-tropical areas of the world. It is found in Central and South America; on the west coast of Africa and in Uganda, Kenya, Tanganyika and Madagascar; in Assam, Burma, Ceylon, Thailand, Indo-China, parts of India, southern China and Formosa; in the Malay Archipelago and in Indonesia; and in northern Australia and the islands of the south Pacific. However, widespread as the incidence of the disease still is, much can be done and is being done about it. Those who are concerned with it will be interested in the recently published account of the First International Symposium on Yaws Control held at Bangkok, Thailand, in March, 1952.[1] It is pointed out in the introduction to the symposium that two major advances of the last decade are destined to influence public health programmes directed at the elimination of yaws. The first is the development of a new and greatly improved form of therapy. Ehrlich's dream of a single injection cure has come true, for there is available today—in the newer repository penicillin preparations—a form of treatment that is therapeutically efficacious, negligible in toxicity, low in cost and readily administered to the patient. The second is the advent of international health work permitting direct services to countries—work carried out by the World Health Organization, the successor of the Health Organization of the League of Nations. Together these two advances have made possible a broad frontal attack on yaws, and within the past few years large-scale projects to bring penicillin therapy to the masses of the people have been undertaken with the technical guidance of the World Health Organization and with supplies furnished by the United Nations International Children's Emergency Fund in Haiti, Indonesia, Thailand and the Philippine Islands. It is pointed out that the concept of communicable disease control by mass treatment is a comparatively new one, but only because the means for attaining the end have previously been lacking. Now that a suitable therapeutic agent is available the work has gone forward rapidly and with considerable success.

The First International Symposium on Yaws Control was organized because it was considered that the time had come for those who had had experience with this new and potentially decisive approach to yaws control to review that experience, to share it with others, and through collective consultation and frank discussion to decide on the most appropriate way to effect yaws control in the light of current knowledge. The symposium was developed along the following lines. The first day was devoted to taking stock of the present information on yaws, and the second to a review of the available information on the use of antibiotics, particularly penicillin, in the treatment of yaws. During the next five days consideration was given to the five phases through which a yaws control project usually evolves: first, preliminary analysis of the extent and nature of the problem; second, development of plans of operation suitable for local conditions; third, demonstration, survey and training; fourth, expansion of the project into a mass treatment campaign; fifth, consolidation, in which the gains of the mass campaign are integrated into the permanent health structure of the area concerned. On the last day of the symposium meetings the subject was the role of international organizations assisting governments in their efforts to control yaws. Over 70 health workers from all parts of the world attended the symposium. In the official publication the individual papers appear with a brief account of the subsequent discussion. The purpose of the publication is to extend the knowledge of this work beyond the limited group who attended the symposium and to show the effective work that has been and can be done in this field.

[1] "First International Symposium on Yaws Control, Bangkok, 1952"; World Health Organization Monograph Series Number 5; 1953. Geneva: World Health Organization. 9½" × 6¼", pp. 418, with 32 illustrations. Price: 22s. 6d.

PICTURE FROM CARDIFF.

THE following paragraph by one of its "Peripatetic Correspondents" was published in *The Lancet* of July 18, 1953. It is such a cheery pen-picture of the Annual Representative Meeting of the British Medical Association that members of the Australian Branches of the Association should be made aware of it.

> A long agenda is tricky to follow at any time, but during a Test Match it's downright impossible. The chairman at Cardiff recently rattled off the figures easily enough: the agenda, then the scores (a note of Arlott helps the experienced ear), then a few votes that somebody's cast, the scores again, and back to the agenda. But the poor chaps down below get quite bewildered, and that young lady with her scoreboard may tell them where they are on the agenda, but it's a scoreboard all the same. And just to scatter the thoughts Old-Traffordwards all the quicker, look at that friendly Australian face, with the quizzical half-smile, on the platform. Give it a green cap with a jutting peak and it's uncommonly like Sir Donald. Which reminds me that there's some comfort in being able to confuse the Australian score with item 133.

Members of the Branches should have no difficulty in recognizing the identity of the "friendly Australian face", especially as the General Secretary of the Federal Council is now in England.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Streptococcus Polysaccharide.

WILLARD C. SCHMIDT (*J. Exper. Med.*, January, 1952) made studies on the preparation, chemical composition and cellular localization after intravenous injection into mice of group A streptococcus polysaccharide. He believed that a study of the fate of immunologically active components of group A streptococci might help to elucidate the relationship of streptococcal infection to the rheumatic process. He endeavoured to devise a mild method for preparation of the polysaccharide to reduce the risk of polymerization, and this was achieved by pepsin digestion of ground streptococcal cells and a careful purification process, either by electrophoresis or by the use of ion-exchange resins. Approximately 0·1% of the starting material was recovered as purified polysaccharide. Chromatographic analysis and reducing values suggested that the polysaccharide contained rhamnose and glucosamine in the proportion of 5:2. Antibody was prepared by injecting rabbits with whole group A streptococci, the globulin fraction obtained and conjugated with fluorescein isocyanate. Mice were injected with 10 milligrammes of polysaccharide preparation dissolved in normal saline and sacrificed at varying intervals, and blocks of tissue fixed in cold acetone containing anhydrous calcium sulphate imbedded in paraffin were used to cut sections, which could be treated with the labelled antibody and examined under the fluorescence microscope. It was found that in mice killed seven minutes after injection, this polysaccharide was present in the tubular epithelial cells of the kidney, but not within their nuclei. After fifteen minutes this was much less, and it had disappeared in thirty minutes. Very rarely could a specifically stained cell be seen in lung, spleen, parenchyma or renal glomerular cells. Precipitin tests performed on mouse blood and urine at the time of autopsy gave positive reactions at a dilution of 1:500 and 1:50,000 respectively. The rapid excretion of this polysaccharide in mice is shown by its absence from body cells in general and its disappearance from the renal tubular cells during the excretory phase.

Rheumatic-Like Lesions in the Rabbit Heart.

L. KIRSCHNER AND J. B. HOWIE (*J. Path. & Bact.*, April, 1952) studied rheumatic-like lesions in the heart of the rabbit, experimentally induced by repeated inoculation with hæmolytic streptococci. This work was an extension of the previous serological work in human patients. They used serological tests and histological examination of tissues in rabbits, and their animals included controls sensitized to horse serum; some of these were desensitized by small doses of horse serum and separately by the administration of antihistamine drugs. The test animals were in two groups: one sensitized by horse serum and injected with killed hæmolytic streptococci group A, the other sensitized by repeated injections of living streptococci producing focal lesions. The rabbits were of a particularly inbred stock maintained over a period of twenty years. In the series in which horse serum was used, a proportion of the animals died in anaphylactic shock with the exception of the histamine-treated series, in which all survived and their histology was normal. Some of the horse serum controls showed experimental serum carditis; the desensitized series showed no lesions. The horse serum sensitized group inoculated with killed streptococci showed severe cardiac lesions in four animals out of six. In the series receiving living hæmolytic streptococci, seven of twelve showed severe cardiac lesions, three showed minor lesions, and two were entirely normal. Blood obtained at the autopsies of all these animals was sterile. Estimation of antistreptolysin, however, showed a correlation between severity of lesion and height of titre; the animals with mild histological effects showed figures of 100 units or less, while the severely affected animals' serum contained as much as 700 units. The authors discuss the varying degrees of lesion seen in the tissue responses in these animals and make comparison with the diseases *periarteritis nodosa*, rheumatic fever, rheumatoid arthritis and disseminated *lupus erythematosus*. They suggest that there is not necessarily a common ætiology, but a variety of anaphylactoid responses to varying antigenic factors.

The Effect of Immune Human Serum on Tubercle Bacilli.

I. BLUHM (*Acta med. scandinav., Supplementum* CCLXXV, 1952) has studied the oxygen consumption of tubercle bacilli mixed with human serum. It was found in 118 experiments out of 130 that less oxygen was consumed in serum from reactors to tuberculin than in serum from non-reactors. It is suggested that this result was due to a bacteriostatic or bactericidal effect of the serum of reactors.

Complement-Fixing Antibodies to Poliomyelitis Virus.

N. GOLDBLUM AND J. L. MELNICK (*J. Exper. Med.*, August, 1952) studied the occurrence of complement-fixing antibodies to type 2 (Lansing) poliomyelitis virus in a normal population of a subtropical area. The tests were carried out on samples of serum from children around Cairo, where the disease is endemic and all children are likely to have been exposed to infection by the age of three years. Second specimens of blood from 16 children were available; these were taken eighteen months after the first. Antigen was prepared from infant mouse brain; it was harvested sixty hours after inoculation, the acetone-ether extraction method of Casals being used. Serum dilutions from 1:4 to 1:16 were tested with this antigen and with normal mouse brain extract as a control. From investigation of 171 sera, it was found that complement-fixing antibody was completely absent when the patients were below the age of one year, although many showed neutralizing antibody. Up to the age of four years, half the children had complement-fixing antibodies; and after the age of nine years the figure fell rapidly. In the tests on paired sera many changes were seen, both in loss and in gain of antibody. Neutralizing antibody titres in the general population rose to a high level by four years of age and did not fall. From observation of the results of both tests, it was possible to identify "old infections", in which complement-fixation findings were negative and neutralization findings positive, and recent infection, in which both were positive; the condition of complement-fixation findings positive and neutralization findings negative did not occur. When the findings from both tests were negative, no infection had taken place.

Differentiation of Corynebacteria.

G. T. COOK AND W. H. H. JEBB (*J. Clin. Path.*, May, 1952) studied a group of starch-fermenting, gelatin-liquefying corynebacteria and their differentiation from *C. diphtheriæ gravis*. They state that the ability of these organisms to produce two toxins, only one of which can be neutralized by diphtheria antitoxin, makes the group significant—they have been isolated from patients with acute sore throat as well as from symptom-free carriers. It was found that all these gelatin-liquefying strains also hydrolysed urea if inoculated onto Christensen's medium and did not reduce nitrates to nitrites; and these last-mentioned tests can be read after overnight incubation, whereas the gelatin-liquefaction may take some days to appear. Pathogenicity tests of the cultures in guinea-pigs must be performed by the intradermal route, when a characteristic ulcerating lesion is produced which is not prevented by 1000 units of antitoxin. If the subcutaneous route used, the guinea-pig may die with the classical appearances, and 500 units will protect the control pig. It is noted that membrane formation is not common in patients infected with these organisms, and it does not appear to spread in epidemics; the isolations have come from single cases. As one of its toxins can be neutralized by diphtheria antitoxin, it appears wise to treat the patients as suffering from true diphtheria.

Significance of Poliomyelitis Complement-Fixing Antibody in Man.

J. CASALS, P. K. OLITSKY AND A. B. SABIN (*J. Exper. Med.*, July, 1952) studied the development persistence and significance of type 2 poliomyelitis complement-fixing antibody in man. The antigen was obtained from virus adapted to newborn mice, and was known to react with serum from immune animals and from persons with poliomyelitis. Children convalescent from poliomyelitis had no type 2 neutralizing antibodies, but monkeys convalescent from types 1 or 3 poliomyelitis had no complement-fixing antibodies for type 2; it seemed that human serum might not react specifically. Sera for testing were obtained from patients in whom virus had been recovered and typed, from patients from whose sera neutralizing antibodies for type 2 virus were absent, from patients with poliomyelitis of unknown strain, and from controls without history of poliomyelitis. Of 13 poliomyelitis patients from whom type 1 virus was recovered, Lansing neutralizing antibodies were present in five and absent in eight. Complement-fixing

antibodies, however, were present in all except one and persisted up to three months in ten. In some the titre was unchanged, in some it was high and then fell. There was no evidence, however, that in these patients type 2 virus had caused the illness. In the second group of patients with poliomyelitis, three-quarters showed titres of over 1:16 during the acute phase, and this lasted into the convalescent phase. There was no significant difference between those who had neutralizing antibody and those who had complement-fixing antibody. In all these patients the titres in later serum samples fell steadily up to ninety days, the period of the tests. In the controls, low titre neutralizing antibody is widespread, up to 85% by the age of fourteen years showing its presence. Complement-fixing antibody, however, was at a lower level in a smaller proportion of subjects, and the titres were lower in winter than in summer. The authors believe that their evidence suggests that infection with type 1 poliomyelitis virus can lead to increase in complement-fixing antibody against type 2, and wish for more evidence in relation to antibody levels in patients when tested against type 1 and type 3 antigen before a useful evaluation of the present experiments can be made.

Hæmolytic Activity of Bacterium Coli.

J. BAMFORTH AND J. A. DUDGEON (*J. Path. & Bact.*, October, 1952) studied the hæmolytic activity of *Bacterium coli*. They found that organisms which produced a small and irregular greenish change on agar plates enriched with 5% oxalated human blood would usually type human red cells added to cultures of the organism in a liquid medium consisting of 1% of peptone in either 0·5% or 0·85% sodium chloride solution made from tap water. If doubly distilled water was used, no hæmolysis occurred. If minute traces of calcium were added, hæmolytic activity appeared. The rate of occurrence of hæmolysis was found to parallel the rate of growth of the culture, and it was also related to the optimum growth temperature. If the culture without added blood was centrifuged and the supernatant added to red cells, no hæmolysis occurred. It appeared clear that the lysis of the red cell depended on the presence of the organism itself and that there was no evidence to suggest the formation of a filterable or soluble hæmolysin by the strains of *B. coli* used in this work.

HYGIENE.

Thermal Destruction of Bovine Tubercle Bacilli.

H. BUNDESEN, T. DANFORTH, H. WOOLLEY AND E. LEHNER (*Am. J. Pub. Health*, February, 1953) report the results of a series of experiments to determine the time and temperature requirements for the thermal destruction of *Mycobacterium tuberculosis* var. *bovis* in chocolate drink, 12% milk and 12% ice-cream mix. The technical procedure and the methods of control are described. The results show that the strain of *M. tuberculosis* var. *bovis* used failed to produce tuberculosis in guinea-pigs after it was exposed to temperatures of 160° to 180° F. for periods of fifteen to thirty seconds in chocolate drink, 12% milk and 12% ice-cream mix.

Cleaning of Various Eating Surfaces.

G. RIDENOUR AND E. ARMBRUSTER (*Am. J. Pub. Health*, February, 1953) describe a method of testing the effectiveness of various methods used to remove organisms from the surfaces of eating utensils; they state that this will give more precise information than can be obtained by the swab-rinse method. Radioactive phosphorus-tagged bacteria were used to coat the surfaces of glass, china, steel, plastic and aluminium eating utensils. The total radioactivity of the bacteria that remained on the surfaces after cleaning was then measured with a specially designed counting apparatus. This was also shown on autoradiographs. From the results obtained the following conclusions were made. Some types of organisms may be so strongly absorbed on some surfaces that normal methods of dish-washing may fail to remove more than a small fraction. The strength of absorption varies with the type of organism. Some are lightly absorbed. Others are strongly absorbed. Absorption of the organism is specific for a surface. Of the bacteria studied, *Micrococcus aureus* was most difficult to remove from a given surface. *Escherichia coli* was the easiest to remove. Percentage removal of *M. aureus* in the absence of soil from china, glass and steel surfaces, with standard detergency and temperature conditions, fell between 97 and over 99. Under similar conditions, percentage removal of the same organism from plastic and aluminium surfaces was between 56 and 84. With plastic eating surfaces, a large variation existed among the different types. This variation did not exist between the china or glass surfaces. With *E. coli*, in the absence of soil, the ease of removal increased for all the surfaces. The percentage removal efficiency varied between 99 and just under 100 for china, glass and steel and between 90 and 98 for plastic and aluminium. When the bacteria were mixed with an organic soil, the efficiency of removal increased greatly for all surfaces. When the bacteria were overlaid with the organic soil, efficiency of removal from plastic surfaces dropped considerably in relation to china, glass and steel. When plates were worn or scratched, removal efficiency decreased for all surfaces, but the same relation existed between different surfaces as with the unworn surfaces. In respect to bacterial film build-up from repeated bacterial soiling and washing, the plastic surfaces exhibited a soil build-up many times that of china before saturation was reached.

Bacteriophage and the Toxigenicity of Corynebacterium Diphtheriæ.

E. PARSONS AND M. FROBISHER (*Am. J. Pub. Health*, March, 1953) report the results of further investigation of the effect of bacteriophage on the toxigenicity of *Corynebacterium diphtheriæ*. A summary of previous work indicates that virulent strains of *C. diphtheriæ* rarely if ever lose their virulence under laboratory conditions. Avirulent strains, however, under certain conditions can become virulent as a result of contact or infection with specific bacteriophage. The authors show that rate of change is affected by pH and temperature and that not every phage affects every culture. The change appears to occur with difficulty or not at all in most strains, and so far only in *mitis* and *mitis*-like strains. All phage-derived toxigenic clones are obviously lysogenic. The number of toxigenic cells developing in avirulent strains in contact with phage far exceeds that normally observed. On some occasions what appeared to be bacteria-free bacteriophage suspensions have yielded typical virulence test results in rabbits. The reactions seem to be due to toxin, but the origin of the toxin is not clear. The authors conclude that as a consequence of these findings the present entire outlook on the natural history and epidemiology of diphtheria must be altered.

Familial Spread of Sonne Dysentery.

R. LEADER (*Med. Officer*, March 7, 1953) investigated an epidemic of Sonne dysentery that involved 615 persons, to determine the mode of spread. Bacteriological examination of 34 samples of different kinds of food for dysentery bacilli yielded negative results. This eliminated the bulk contamination of food as a source of infection. Other members of a family in which one or more members were found to have dysentery were then investigated; one symptomless carrier was found to every 2·4 persons with symptoms of dysentery among the "positive cases". In the age group sixteen years and over the ratio was 1:1·4; in the age group ten to fifteen years the ratio was 1:1·9; in the age group five to nine years the ratio was 1:3·8; in the age group nil to four years the ratio was 1:3·9. Infected adults were frequently symptomless carriers, while infected children usually developed symptoms. In families in which a case was reported and the other members were then investigated, it was found that in the under sixteen years age group 74% of members were "positive" and in the over sixteen years age group 35% were "positive" for dysentery bacilli. The author concludes that dysentery is almost invariably a family infection involving the majority of members of the family.

Immunological Studies of Poliomyelitis and Rabies Viruses.

A. MILZER AND F. NICHOLSON (*Am. J. Pub. Health*, April, 1953) studied the immunological relationship between the poliomyelitis and rabies viruses. In a series of three active immunity experiments no evidence of cross-immunity was detected between mice vaccinated with an ultra-violet-inactivated poliomyelitis (Lansing) or rabies virus vaccine. Furthermore, no evidence of cross-immunity was found between the poliomyelitis and rabies viruses in neutralization tests with hyperimmune poliomyelitis and rabies antisera. On the other hand, in every experiment the vaccine or hyperimmune serum tested provided significant protection against the homologous virus. The authors state that the results obtained in both active and cross-neutralization tests indicate that there is no significant immunological relationship between the Lansing strain of poliomyelitis virus and the rabies virus. These experiments do not support the theory that rabies vaccination is of value in prophylaxis against poliomyelitis.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LXXIV.

THE INDICATIONS FOR SYMPATHECTOMY.

HUNTER AND ROYLE in a brilliant though unsuccessful attempt to alleviate spasticity focused attention on the sympathetic nervous system as a hitherto unexploited surgical field. There resulted an energetic and optimistic mutilation of this system in the attempted alleviation of widely diverse infirmities. An experimental trial of different techniques on these many disorders has resulted today in a standardized surgical approach for a set of conditions now well attested as likely to be benefited.

Peripheral Vascular Disorders.

The most constant and outstanding benefit of sympathectomy is found in the treatment of the various vascular diseases of the limbs. At first used only in vasospastic disorders such as Raynaud's phenomena, it was found later to be beneficial in that ill-defined group of a usually unconfirmed pathology known as Buerger's disease. It was later realized that sympathectomy had a place in the treatment of all obliterative vascular diseases, in the diabetic and senile, and in the acute occlusive emergencies. The view is now generally taken that in any peripheral vascular disturbance, sympathectomy must be seriously and often urgently considered.

Such disorders present themselves either superficially as a vascular deficiency, evidenced by the signs or threat of gangrene, or as muscular ischæmia evidenced by claudication.

The absolute indications for surgery in these conditions are not to any extent dependent upon pre-operative tests. If vasorelaxant drugs such as "Etamon" have an obvious good effect, then the indication is absolute; if a test "Novocain" block of the appropriate sympathetic chain is effective, the indication is again strong. On the other hand, however, it is not wise to assume that a negative result of these tests is a contraindication to surgery. Sympathectomy may be very successful even though "Etamon" may give no relief. Sympathetic injection may be ineffectual, and it must be remembered that proper and adequate "Novocain" infiltration cannot always be surely assumed.

The indications for sympathectomy in all types of peripheral vascular diseases are wide. It may greatly modify the whole pattern of treatment even when alone it is inadequate to save a limb.

It must be considered in acute vascular emergencies, such as traumatic arterial spasm or the crushing of a limb, when the site distal to the injury appears to be deprived of adequate circulation. The same indication applies to acute embolism or thrombosis which cannot be, or has not been treated by direct attack.

In the vasospastic disorders it must be used and is our only remedy, even though we are aware of the possibility of later return of the trouble, for it must be remembered that after a lapse of years the sympathetic system has a way of reasserting itself. In obliterative arterial disease, it should be used as soon as the circulation is known to be inadequate. It must not be delayed until actual gangrene has begun to develop. If such delay has occurred, sympathectomy should still be performed. This applies to all ages, and is in no way contraindicated by an extensive main arterial involvement. It may completely arrest the condition and lead to healing of the gangrenous areas, or it may enable conservative surgery to be used at a later date. Its value in this way is very great. It often abolishes the terrible pain which otherwise forces the surgeon to early amputation. The stay in proceedings which is thus gained enables demarcation to occur, and later very gentle and very conservative removal of the gangrenous parts may be successfully performed, or a simple below-knee amputation may win the prize of a useful joint. In this way the ablation of the whole limb may be avoided. Immediate amputation of the lower limb through the thigh in the treatment of a gangrenous toe should now rarely be necessary.

Sympathectomy has been found to give dramatic benefit in peripheral venous thrombotic conditions, and the swollen white leg, even though of long standing, may subside like a pricked balloon after this operation.

In conditions which might be termed vascular disorders of neuropathic limbs, such as the cold atrophic extremity which may follow poliomyelitis, sympathectomy is of great value in restoring warmth and in the prevention or healing of ulcers.

Patients with local circulatory disturbances caused by chronic congestive ulceration which resists all usual treatment may be greatly benefited by sympathectomy. In some such cases in which plastic repair appears to be necessary, sympathectomy will prepare the ground for successful plastic grafting.

When claudication alone is the complaint, sympathectomy from the first to the third lumbar segments should be performed. It is very likely to lead to considerable improvement, particularly in the less severe cases. In more severe cases it may later require to be supplemented by a tentotomy of the *tendo Achillis*.

It must be remembered that such conditions as coronary insufficiency or cardiac dyspnœa make the operation unwise because of the resultant drop in blood pressure.

Intractable Pain.

Sympathectomy has been used in the attack on various types of intractable pain and may now be regarded as standard and effective treatment for *angina pectoris*, some limb causalgias and intractable abdominal pain. The sufferer from angina is not a surgeon's dream, but adequate sympathectomy can usually be carried through safely enough. Stellate ganglionectomy, supplemented if necessary and later by sympathectomy of the dorsal chains as low as the sixth thoracic level, may give untold relief to these patients, and in the hands of a competent surgical team the risks are well justified.

The use of sympathectomy should never be forgotten in producing relief from intractable painful conditions in the abdomen, as for instance in pancreatic calculosis or in inoperable newgrowths or ulcers, and other conditions which in the absence of demonstrable pathology must be regarded as dyskinesias. I have had very happy experiences with such operations.

Some limb causalgias respond remarkably to sympathetic block or section. The causative injury often may have been trivial, but the resultant painful, hypersensitive and discoloured limb is most distressing. Such disordered physiology, however, appears to operate from a high neurological level, and it may occasionally be the unhappy experience of the surgeon, having abolished the causalgia or vasomotor disturbance in the injured limb, to be confounded by the sudden appearance of a similar condition on the other side.

The painful phantom limb is a distressing complaint which is likely to make the enterprising neurosurgeon climb the tree of the nervous system in a series of pain-interrupting operations, even to cortical level. Sympathectomy should therefore not be advocated confidently, but it may be successful and is worthy of trial, particularly if performed early in the complaint.

Sometimes a hypersensitive and swollen area remains at the site of a severe fracture. After the fracture has united this may linger as a major disability. In such cases sympathectomy may have a charmed action.

In severe pelvic pain, due either to physiological disorders of menstruation or to inoperable malignant disease, extirpation of the pre-sacral nerves has been employed. It is not effective in the relief of pain from malignant disease involving the pelvic organs and parietes.

Hypertensive Disease.

In 1934 Adson and Pete conceived the idea of lowering hypertensive blood pressure by thoracic sympathectomy and splanchnicectomy. The intervening years have seen a wide trial of the original operation and its various modifications. The enormous benefits conferred on a very considerable proportion of sufferers leave no doubt of its usefulness in the general attack on the problem of hypertensive disease. The indications are not easily summarized; but it may be said that for patients under fifty years of age who suffer from symptoms of the disease, the operation should receive very serious consideration.

Hyperhidrosis.

The sympathetic system subserves the function of sweating. In fact, study of the sweating pattern is the only way of assessing the extent of a sympathectomy. The use of sympathectomy in combating hyperhidrosis of an

extremity is therefore obvious. The condition is fortunately not common, but when it occurs in the hands, hyperhidrosis can wreck a life and throw its victim shrinking out of normal society. In such cases, appropriate sympathetic section confers an inestimable boon. The patient can with confidence shake hands.

Cerebral Thrombosis.

In recent years favourable reports have directed the attention of both medical and lay readers to the possibility that sympathetic interruption may improve the state of patients suffering from presumed cerebral thrombosis.

It is essential before undertaking such treatment for the surgeon to be reasonably sure of the diagnosis. Cerebral tumour and cerebral abscesses, as well as intracranial hæmatomata, are only too often classed as "cerebral thrombosis". It may therefore be wise for the surgeon, if in any doubt, to perform cerebral angiography, both to exclude other conditions and to seek further light on the nature of the vascular accident. This having been done, sympathetic interruption by "Novocain" should be tried. If this is done in the early days after the disaster, it may have a most dramatic effect.

Other Conditions.

Sympathectomy has been tried empirically in many conditions of obscure nature and difficult to relieve. Good claims are made from some quarters for its use in labyrinthine vertigo, the rationale being a supposed induced hyperæmia in an ischæmic labyrinth. A successful "Novocain" block (evidenced by a Horner's syndrome) made during an attack is the indication for the operation.

In some cases of migraine-like headache likewise, a successful "Novocain" block may point the way to more permanent relief by means of sympathectomy. Most cases of migrainous nature do not, however, react favourably to this test.

Summary.

To summarize the indications for sympathectomy, it may be said to have great value in practically all peripheral vascular disorders, whether they are organic and obliterative or neuro-vascular in origin. It has value in the relief of visceral pain, both thoracic and abdominal, it can revolutionize the life of a hypertensive invalid, and it may confer great benefit in various conditions of sympathetic overactivity such as hyperhidrosis. Its trial in various other conditions of obscure pathology is occasionally rewarded.

DOUGLAS MILLER,
Sydney.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on April 23, 1953. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital. Part of this report appeared in the issues of July 25 and August 1, 1953.

Popliteal Embolectomy.

DR. T. F. ROSE showed a woman, aged sixty-seven years, who had been treated for three years for hypertensive congestive cardiac failure with attacks of fibrillation and œdema of the legs. Treatment was with digoxin and mersalyl. On April 6, 1953, she had had sudden severe pain behind the right knee, spreading down the leg to the toes, with numbness and paralysis of the leg. The leg below the knee was found to be cold, white and paralysed, and sensation was absent. The thigh was normal. Femoral pulsation was felt below the inguinal ligament, but pulsation of the popliteal artery and of the arteries of the ankle was absent. One hour later the leg was cold and mottled blue, and the medial side of the thigh up to the groin was cold and mottled with loss of sensation. It was decided that the embolus must have lodged in the femoral artery at the point where it gave rise to its deep femoral branch. Operation was performed immediately, the femoral artery being exposed in the femoral triangle. The artery was pulsating vigorously above the deep femoral origin but not below. However, while the artery was being mobilized, that segment commenced to pulsate also, and it was obvious that if an embolus had lodged at the origin of the deep femoral artery, it had moved distally. Accordingly the popliteal artery with the origin of its branches, the anterior and posterior tibial vessels, was exposed. The artery was pulsating down to a point one inch above its bifurcation. Below that point pulsation was absent and the vessel felt very hard. Arteriotomy was performed, and the clot was found and removed. It appeared to be old clot. It was noticed that the arterial intima was smooth. Anticoagulants were administered, and the pain did not reappear when the patient recovered from anæsthesia. The mottled blue appearance of the limb disappeared in a few hours, and sensation and muscle power returned on the following day, but the leg did not become warm until forty-eight hours after the operation. On the second day a large hæmatoma appeared in the femoral wound and had to be removed. Anticoagulant therapy was thereupon suspended, and subsequent convalescence was uneventful.

Infected Dentigerous Cyst of the Mandible.

In conjunction with DR. JOHN BAIRD, Dr. Rose showed a woman, aged twenty-seven years, who in October, 1952, had undergone a mandibular nerve block for extraction of a carious first left lower molar tooth. One week later she developed pain and swelling of the left side of the face, associated with difficulty in opening the mouth due to stiffness of the left mandibular joint. Penicillin therapy had no effect, and the condition became worse. Two discharging sinuses developed, one opening into the mouth and one into the face in front of the anterior border of the masseter muscle. The discharge appeared to be sterile. She then noticed that as her face became more swollen, her left lower lip and the gum and teeth of the left side of the mandible became numb. In January, 1953, an abscess was incised where it was pointing on the cheek near the sinus, but the condition improved little. In February, 1953, the condition flared up, and the patient was sent to the Royal North Shore Hospital of Sydney, with a diagnosis of recurrent osteomyelitis of the jaw. She had a raised temperature and a large swelling on the left side of the face, with a depressed discharging sinus in front of the swelling and severe trismus. The left side of the chin, the left lower lip and the teeth and gum of the left side of the mandible were numb. X-ray examination showed a large dentigerous cyst arising from a malplaced third molar tooth and involving both the horizontal and ascending rami of the mandible. Infection of that cyst was the obvious cause of the patient's condition. As penicillin therapy had no effect, the outside sinus was dilated and was found to lead to the ascending ramus of the mandible, on the lateral wall of which most of the bone was missing. The manœuvre released much pus. Tissue removed from the cyst appeared to be granulation tissue only, and from it only a coagulase-negative *Staphylococcus albus* was grown. Even while the patient was under the anæsthetic, the trismus remained the same; and although later her general condition improved and her temperature became normal, the swelling persisted. Early in March an approach was made to the mandible through an incision rounding its angle. A large dentigerous cyst full of infected granulation tissue was removed, together with all its wall and the offending wisdom tooth. The cyst was found to be invading the masseter, temporalis and internal pterygoid muscles, which explained the persistence of trismus. In three weeks' time the wound was well healed, the sinus had closed, the trismus had gone, and the face was practically normal apart from the puckered scar of the sinus, which would have to be rectified by plastic surgery. Dr. Rose said that the case illustrated the value of dental X-ray examination in all cases in which teeth had to be removed. X-ray examination of the mandible must be of good standard, so that mistakes in diagnosis would not be made. In the present case, the condition had not been diagnosed until correct X-ray examination was made at the hospital.

Neurofibrosarcoma of the Leg.

In conjunction with DR. K. J. FAGAN, Dr. Rose showed a man, aged thirty-five years, who had been admitted to hospital in November, 1951, with a history of a swelling at the back of his left leg just above the ankle for three years. It was growing very slowly, but had only recently caused inconvenience in the form of downward-shooting pain. The swelling was smooth, hard and non-tender, and situated behind the tibia and fibula just above the ankle joint. It was unattached to bone or skin. X-ray examination showed it to be calcified. Operation disclosed that it was a solid tumour arising from the posterior tibial nerve. It was well

encapsulated, except at one point where it was invading the belly of the *flexor hallucis longus* muscle. The tumour, together with the neuro-vascular bundle and the involved muscle, was excised. Dr. Graham reported that the main microscopic appearance was that of a neurofibroma of benign type in spite of its infiltrative powers. Nine months later, the patient noticed some painless hard skin nodules near the operation scar. Those were removed, and on examination were reported as being sarcomatous. He then noticed that the swelling deep in the leg was recurring very rapidly, and he was readmitted to the Royal North Shore Hospital of Sydney. A large infiltrating mass was present above the ankle in the posterior compartment of the calf, fixed to the deeper structures in the skin but not to the bone. It was beginning to ulcerate the overlying skin, and both sensory and motor nerve changes had occurred. X-ray examination showed no involvement of bone in the leg, but metastases were scattered over the lung fields. It was decided to amputate the limb palliatively through the thigh. Pathological examination showed that the condition was undoubtedly sarcomatous, and the outlook for the patient was hopeless.

Carcinoma of the Stomach.

DR. E. F. LANGLEY discussed the case of a woman, aged sixty-two years, who had been admitted to hospital in February, 1950, with a history of "gastric flu" eight weeks previously and "soreness" in the epigastrum. Since then, she had experienced a feeling of weight in the upper part of the abdomen half an hour after some, but not all, of her meals. Specific interrogation revealed few other significant symptoms apart from occasional vomiting, the vomitus being blood-streaked on one occasion only, black motions since Christmas, 1949, and the loss of one stone in weight in eight weeks. The patient was obviously wasted and had a tender, non-movable mass palpable in the epigastrium. After blood transfusion, laparotomy was performed, and a large carcinoma was found involving the pylorus, antrum, lower third of the stomach and left lobe of the liver. It was removed completely and gastro-jejunostomy performed. The patient remained well till December, 1952, when she was killed in a motor accident. The lesion was found microscopically to be a carcinoma directly involving the liver.

Carcinoma of the Transverse Colon.

Dr. Langley's second patient, a woman, aged forty-nine years, had undergone transverse colostomy in October, 1952, for increasing intestinal obstruction with satisfactory relief of her symptoms. Five weeks later, having been treated with quinidine for ventricular extrasystoles, she underwent laparotomy, and a carcinoma of the transverse colon was found adherent to a loop of jejunum and to part of the greater curvature of the stomach. This was removed, and the colon was closed. The colostomy opening closed within a few days, and the patient's condition remained satisfactory. The pathologist reported that the tumour was a carcinoma producing mucin, and that it probably arose from the colon.

Common Iliac Artery Thrombosis.

Dr. Langley then showed a man, aged fifty-six years, who had been treated medically two and a half years previously for severe pain in the right foot and back of the calf while walking, and had been free of pain for eighteen months. More recently, sharp pains had occurred in the left foot and calf and were aggravated by walking and relieved by rest. For three days before his admission to hospital the patient had had severe pain below the left knee and a red swelling on the left foot. His left foot and ankle were red, swollen and œdematous, the *dorsalis pedis* and posterior tibial vessels were not palpable in either foot, and left femoral pulsation was absent. Aortic arteriography revealed a thrombosis of the right iliac artery; retrograde arteriography revealed that the left femoral artery had failed to fill with opaque medium.

Dr. Langley's last patient, a man, aged sixty-five years, had since 1947 had pain in the right hip and buttock passing down the right leg and foot and causing numbness. It had gradually become worse, causing considerable difficulty with walking. It was aggravated by cold and relieved by heat. Pain had also been present in the left leg below the knee for twelve months. Pulsation of the femoral and posterior tibial arteries was absent on the right side, and that of the *dorsalis pedis* artery on both sides. Aortic arteriography revealed right common iliac artery thrombosis.

Prolonged Survival After Gastro-Œsophagectomy for Cancer.

DR. F. F. RUNDLE referred to two patients who had been previously shown at the clinical meeting held at the Royal North Shore Hospital in July, 1951. He said that he presented the progress reports because final assessment of the palliative value and long-term results of the operations concerned required accumulated data.

The first patient, a woman, aged sixty years, had undergone total gastrectomy on March 5, 1951, for a carcinoma involving almost the entire length of the lesser curvature of the stomach and invading the lymph nodes extensively. She had since remained fully active, in good health and free from pain. Her weight was stationary. Clinical, radiological and œsophagoscopic examination showed no evidence of recurrence. She did, however, complain of symptoms of œsophageal reflux. She had heartburn and reflux of bilious fluid into the mouth when she lay on her right side or when she stooped. Occasionally the heartburn and reflux woke her from sleep at night, but they had been promptly relieved by raising the head of the bed on twelve-inch blocks.

The second patient had undergone a supraaortic œsophagogastrectomy for advanced carcinoma of the mid-thoracic part of the œsophagus in September, 1950. She still survived, but during the past year longstanding unrelated personality changes had worsened and her local practitioner reported that she was verging on a psychotic state.

Biliary Fistula with Electrolyte Loss and Deficiency.

Dr. Rundle then showed a woman, aged sixty-seven years, who had complained of severe flatulence during 1950, culminating in an attack of acute cholecystitis in October. Relief of pain and toxic symptoms followed cholecystostomy, when stones were removed and the gall-bladder was drained. A fistula persisted after removal of the tube, except for a two-month period early in 1951. A severe attack of pain was followed by the discharge of many small stones six months after the cholecystostomy. In December, 1952, she suffered severe continuous epigastric pain for two days and then became jaundiced for three weeks with pale stools and dark urine. She lost seven pounds in weight while she was jaundiced. On her admission to hospital she had no jaundice, and the serum bilirubin content was 0·9 milligramme *per centum*. She had slight evening pyrexia and tachycardia, and her weight was eight stone thirteen and a half pounds. She underwent operation on February 2, 1953. After separation of dense adhesions (mainly of the colon) to the anterior abdominal wall and gall-bladder, cholecystectomy and choledochostomy were performed. A probe was passed down to the region of the ampulla of Vater, and alongside its tip a small stone could be felt. It was removed by duodenotomy and meatotomy of the ampulla of Vater. A T-tube was sutured snugly into the common bile duct, the lower limb of the T passing through into the duodenum. No complications followed. After the operation the total electrolyte loss in the bile was measured daily as well as that in the gastric aspirate. It was found that during the five days immediately after operation a total of 464 milliequivalents of sodium, potassium and chloride was aspirated from the stomach as compared with a total of 452 milliequivalents of the same electrolytes drained in the bile through the T-tube. The daily volumes of gastric aspirate averaged 465 mils, a high normal figure. Dr. Rundle said that the data were presented to emphasize a point sometimes overlooked by surgeons that the electrolyte loss through a biliary fistula was comparable to that lost with continuous gastric aspiration. It might therefore lead to severe constitutional symptoms.

The next patient, a man, aged sixty-two years, had suffered from hypochloræmia, alkalosis and extrarenal uræmia in the course of surgical treatment for gall-stones. He had been admitted to another hospital on November 20, 1952, suffering from obstructive jaundice, and had undergone cholecystectomy and removal of stones from the common bile duct. After the operation he failed to thrive. After the catheter was removed from the common bile duct, he continued to drain all his bile through the wound. In addition he occasionally vomited small quantities. Mental depression, extreme muscular weakness, circulatory collapse, and drowsiness verging on semicoma were prominent clinical features of his electrolyte imbalance. On December 23 biochemical studies showed that there was no chloride in the urine, that the serum chloride content was 490 milligrammes *per centum*, the blood urea content 190 milligrammes *per centum* and the carbon dioxide combining power 79 volumes *per centum*. When a stomach tube was passed, much gas escaped under pressure and 23 ounces of fluid were promptly aspirated. Contributory factors to his electrolyte upset were an inadequate diet, the biliary fistula and a moderate degree of gastric dilatation. Clearly hypochloræmia was ultimately the chief disorder. Intravenous saline therapy corrected the deficiency and produced a dramatic improvement in the clinical condition. His pulse rate fell from 140 to 100 per

minute overnight, and he felt stronger and clearer mentally. After forty-eight hours he was able to ingest all his sodium, potassium and chloride requirement. Salt reappeared in the urine; the serum chloride level rose to 585 milligrammes *per centum* and the carbon dioxide combining power fell to 60 volumes *per centum*. Successive readings of the blood urea value were 190, 95, 62 and 33 milligrammes *per centum*.

Problems in Common Bile Duct Surgery.

Dr. Rundle showed three patients to illustrate problems in common bile duct surgery.

The first problem was one of diagnosis. The patient was a man, aged seventy-two years, who stated that during the six years up to 1946 he had frequently suffered from burning epigastric pain after meals relieved by alkalis and by vomiting. This then passed off and he remained comfortable until one month before his admission to hospital in January, 1953, when he developed severe heartburn, epigastric pain and nausea after meals. The epigastric pain was partly, but not completely, relieved by vomiting; alkalis gave no benefit. The pain came on immediately after food and was located across the upper part of the abdomen; it was more or less constantly present. His appetite remained good, but he was afraid to eat and lost half to one stone in weight during the month. Ten days before his admission to hospital he noticed that his skin and eyes were yellow; his urine was dark and his stools were pale. He was not sleeping well and vomited frequently at night. The vomitus was copious and contained undigested food. He was noted on examination to be an elderly man in obvious distress with jaundiced skin and sclera. Tenderness and guarding were present in the epigastrium and both hypochondria. The liver edge was palpable. One observer thought that he could feel an epigastric mass. For one week after admission to hospital he vomited frequently and in large amounts, the vomitus containing undigested food; it was not conspicuously bile-stained. His jaundice then gradually decreased, and he felt better. On January 19, 1953, the abdomen was opened through a mid-line upper abdominal incision and extended as an "L" to the right below. Much fairly recent plastic peritonitis was found involving small and large bowel. No lesion was found on careful palpation of the stomach and duodenum. The pancreas was enlarged and firm. Multiple areas of fat necrosis were visible in its surroundings. The gall-bladder was grossly diseased and contained many stones. The common bile duct was dilated and its wall thickened. It contained four or five large stones just above the level of the duodenum. Cholecystectomy, choledochostomy and removal of stones were carried out with transduodenal dilatation of the ampulla of Vater and irrigation of the common duct. T-tube drainage of the common duct and Levine tube drainage of the duodenum were instituted. His post-operative course was uneventful, and he was discharged from hospital on February 11. Dr. Rundle said that it was concluded that the intense epigastric pain which dominated the patient's history was due to pancreatitis, of which there was ample evidence at operation.

The next case was one of a small stone very firmly impacted in the ampulla of Vater and requiring trans-duodenal removal. The patient, a man, aged forty-eight years, gave a history of two previous "attacks of gall stones". On each occasion he had suffered severe pain in the right hypochondrium radiating through to the back and associated with severe malaise and vomiting. The first attack had occurred three years previously and the second had occurred one year previously, and each had persisted for many hours. In September, 1952, the pain recurred and again passed off after some eight hours. Two to three days later he noticed that his scleræ were yellow and his urine was dark. Since then also his stools had been pale, and he had lost his appetite and become progressively more jaundiced. Over a period of six weeks his weight decreased by 21 pounds. He had two attacks of shivering with fever and sweating. On October 21 the gall-bladder and bile ducts were explored. Advanced cholecystitis was found. The gall-bladder contained numerous stones and was accordingly removed. The common duct was moderately dilated and thin-walled. From its supraduodenal portion two very small calculi were removed; they were thought insufficient to be causing complete obstruction. A leaden probe could not be clearly demonstrated to pass into the duodenum, and therefore the ampulla of Vater was exposed transduodenally. Its meatus was dilated, and a small calculus was found firmly impacted in the ampulla. Meatotomy was performed to gain access to the calculus. Even so, there was difficulty in extracting it. A T-tube was passed into the common duct, the lower limb of the T entering the duodenum through the ampulla. The patient made an uneventful recovery.

The last problem illustrated was that of reconstruction of the common hepatic duct after operative excision. The patient, a woman, aged twenty-eight years, had been referred from a country town with a biliary fistula in July, 1950. Cholecystectomy had been performed in June, and she had afterwards drained all her bile through the wound. The doctor stated that obtaining adequate exposure had been difficult, and he had inadvertently resected a portion of the common duct near the liver. On her admission to the Royal North Shore Hospital of Sydney her stools were clay-coloured and bile was being discharged copiously through two points in the wound. A cholangiogram prepared after introduction of a catheter into one of the fistulous openings showed that it communicated directly with the hepatic ducts in the right lobe of the liver. Those were dilated, a state which suggested some stenosis of the right hepatic duct. In August, 1950, the lower end of the common duct was identified, after intubation from the duodenum, and traced upwards. Very dense adhesions were encountered. A length of about one inch of the common duct just below the *porta hepatis* had been destroyed or excised. The exit of the right and left hepatic ducts from the liver was identified and dilated with sounds. A Y-shaped "Latex" rubber tube was introduced, the limbs of the Y passing into the two hepatic ducts. The vertical limb was drawn into the lower end of the common duct and on through the ampulla of Vater into the duodenum. About eight inches of tubing lay free in the duodenal lumen. The cut end of the common duct was then carefully sutured to the *porta hepatis* to reconstitute the common hepatic duct. The patient's convalescence was uneventful, but during the first year after the operation she had several transient attacks of malaise and fever. Dr. Rundle said that for the past two years she had enjoyed excellent health, had gained one stone in weight, did a part-time job as well as her own housework (she was married with two small children), and played tennis twice weekly. She had not been jaundiced since the operation.

Sudden Hemiplegia due to Cerebral Tumour.

DR. G. K. VANDERFIELD showed a man, aged fifty years, who, apart from a chronic skin condition, had been well until eight days prior to his admission to the Royal North Shore Hospital of Sydney. He first had a seizure beginning on the left side of the body. In the following days he had further seizures and developed left-sided hemiplegia. Although there was no clinical evidence of raised intracranial pressure, right carotid angiography and pneumoencephalography established the presence of a large space-occupying lesion located in the right fronto-parietal region. The angiographic features suggested that it was a malignant neoplasm. A burr hole was introduced over the suspected site, and a large cystic and solid tumour was encountered on needling of the brain. Immediate biopsy examination of the tumour by Dr. C. S. Graham indiacted that it was a glioblastoma; so no more than aspiration of the cyst was performed, as it was not considered operable. Afterwards the patient was improved and able to return home.

Subarachnoid Hæmorrhage due to Arterio-Venous Malformation of Brain.

Dr. Vanderfield's second patient was a man, aged forty-one years, who had been in a hospital for five weeks in 1945 because of a subarachnoid hæmorrhage. For some days before his admission to the Royal North Shore Hospital of Sydney during 1952, he had had severe left fronto-parietal headache and symptoms suggesting another subarachnoid hæmorrhage. That was confirmed by lumbar puncture. Left carotid angiography revealed a large arterio-venous vascular abnormality in the left hemisphere, filling mostly from an enlarged anterior cerebral artery and draining into the vein of Galen. Active bleeding ceased, but he remained severely disabled with intense headaches and noises in his head. Craniotomy was performed on July 15, 1952, when via a frontal bone flap the distal part of the abnormally large left anterior cerebral artery was exposed before its entry into the malformation and occluded by a silver clip. Recovery from the operation was uneventful. Post-operative angiograms showed non-filling of the anterior cerebral artery and faint filling of the malformation mainly from middle cerebral communications. Dr. Vanderfield said that it was presumed that the procedure had effectively reduced the chances of a further hæmorrhage; it had definitely relieved the patient's symptoms and enabled him to return to his work as a boat-builder.

Subarachnoid Hæmorrhage due to Intracranial Aneurysm.

Dr. Vanderfield also showed a woman, aged thirty-nine years, who had been admitted to the Royal North Shore

Hospital of Sydney in November, 1952, as an "emergency" patient under the care of Dr. F. Lawes. Ten days earlier she had developed severe pain behind the left eye, which became worse, and she lapsed into semicoma just prior to admission to hospital. She was found to have subarachnoid hæmorrhage, which was continuing, as five days later there was still evidence of fresh bleeding on lumbar puncture. Carotid angiography was performed straight away and an aneurysm demonstrated on the distal part of the left internal carotid artery; also the presence of multiple aneurysms was excluded and a good cross circulation via the circle of Willis was indicated by the full studies. In the first instance and as a matter of some urgency, the left common carotid artery was ligated in the neck. That was well tolerated and brought about clinical improvement. One week later, at craniotomy, the internal carotid artery was "clipped" above and below the aneurysm, which was thus excluded from the circulation by "trapping". Some days after operation she developed a transient hemiplegia presumably due to vasospasm; this responded well to stellate ganglion blocks. She was very well on discharge from hospital five weeks after operation, and she had remained symptom-free.

(To be continued.)

AUSTRALIAN ASSOCIATION OF PHYSICAL MEDICINE
(BRITISH MEDICAL ASSOCIATION).

ANNUAL MEETING.

THE eighth annual meeting of the Australian Association of Physical Medicine was held in Sydney on April 14, 1953.

The following items were presented during the course of the meeting. On behalf of Dr. F. Edmundson (New Zealand), Dr. W. G. Rich read a paper on subdeltoid bursitis and its treatment with ultrasonics. Following a paper presented previously, Dr. L. Wedlick gave further experiences with intensity time curves. Details of cases of ankylosing spondylitis in the female were given by Dr. Paul White, and Dr. B. G. Wade gave an account of his experiences overseas, with illustrations of work being done at various rehabilitation centres. Dr. Rich presented a paper on the treatment of sinusitis with ultra-short-wave. Two very interesting films were also shown, one being on "Œsophageal Speech-Training in Laryngectomized Patients" and the other on the "Surgical Approach to the Shoulder Joint".

A clinical afternoon was also held in the Arthritic Clinic at the Royal Prince Alfred Hospital, when demonstrations were given by Dr. S. G. Nelson, Dr. T. W. Burgess and Dr. Reedy.

Election of Office-Bearers.

Office-bearers for the forthcoming year were elected as follows:

President: Dr. P. J. Alpers.

Vice-President and President-Elect: Dr. Selwyn Nelson.

Vice-President: Dr. N. T. Bull.

Secretary-Treasurer: Dr. L. T. Wedlick.

Committee: Executive members and Dr. J. Shanasy.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

PHILOPELISM.[1]

[The Australian Medical Journal, June, 1866.]

IN this part of the world among the many opposites that contrast with what is the rule in Europe we have the curious anomaly of a large majority of the popular branch of the legislature actually fighting with the most determined pertinacity against measures whose sole object is to prevent the injury which European legislatures are so anxious to repair.

The pollution of the rivers of England by admixture with the refuse of dense populations dwelling on their banks has caused an expression of constant regret for many years, even from those whose interests compelled them into an involuntary participation in the evil: and the one great purpose of the sanitary movement has been to purify them even at any cost and any sacrifice. This laudable work is being, however, slowly yet steadily carried out and even the uninformed mind is becoming conscious of the advantages of limpid streams as compared with what practically amounts to an open sewer of more or less diluted fæculence. It has been reserved for the Parliament of Victoria—the colony to which some political economists are in the habit of pointing, as exhibiting the wonderful progress of a new country—to demonstrate the possibility of a legislature or at least a majority of it, doing all in its power to encourage the production of disease by insisting on expunging from the Public Health Bill the clauses relating to the pollution of the stream which flows past this city to the sea. Happily, that much of our Parliament which, in some minds, is associated with an indisposition to perceive great common advantages, has so far preserved us from the disgrace and danger of wilfully defiling the only considerable stream in our colony except that which forms our northern boundary. But the prevention of the evil has been accomplished only by the sacrifice of an otherwise most useful measure. The Council restored the clauses that the Assembly expunged. The Assembly again expunged them and the Council very properly has refused to pass the bill without the restoration: and thus it happens that for the sake of gratifying their selfish and filthy instincts the majority of the Assembly has allowed an excellent measure to become inoperative. On the ground of public safety we protest most strongly against the base and scandalous motives in obedience to which our legislators do not scruple to sacrifice the general good, for the sake of encouraging some contemptibly insignificant manufacturing interests: but while protesting we in no wise wonder at such a result.

It is neither our want nor our desire in this journal to touch upon political subjects, simply as such, but we do not think it at all beyond the scope of our functions to deprecate that democratic proclivity, which has, unfortunately for the community, sent into our legislature men whose habits and education render them utterly incapable of perceiving the inestimable advantages of preventing our rivers from becoming sewers and who are obviously unaware of the determined efforts that have been made of late years, in the old country, to counteract the indifference of our forefathers in this respect.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

THE USE OF PARALDEHYDE IN ALCOHOLIC DELIRIUM TREMENS.

SIR: It was with great interest and considerable astonishment that I read Dr. D. G. Steyn's condemnation of the use of paraldehyde in alcoholic *delirium tremens* (M. J. AUSTRALIA, July 18, 1953).

For many years paraldehyde has been the sheet-anchor of sedation for "D.T.'s" at this hospital for the two very good reasons that it is (i) effective and (ii) safe.

To be really effective it must be given in adequate dosage. To give doses of one or two drachms is merely playing with the problem and may well give rise to the accusations that it "acts as a cerebral excitant in alcoholics". Standard procedure here is to give, as an initial sedative, a dose of "Godfrey's mixture" (named after the Superintendent of the Receiving House, Royal Park, who advocated the prescription a generation ago). It contains paraldehyde, four drachms, chloral hydrate and potassium bromide, thirty grains each, and once included, as a tribute to the pharmacology of the past, tincture of digitalis, ten minims, "to strengthen the weakened action of the heart". The old instruction was to give this dose with brandy, two ounces, and repeat in two hours with brandy, one ounce, if the patient were not asleep. It has never been recorded that a "repeat" was necessary, and in this austere age even the brandy and digitalis are omitted with no noticeable decrease in effect.

Now it is quite true that some unexpected deaths occur in any group of heavy alcoholics within the first twenty-four to forty-eight hours of admission to hospital, but there is not the least evidence that these are due to any toxic

effect of paraldehyde. The three principal causes are Wernicke's encephalopathy with steady development of increasing depth of coma, sudden death with acute cardiac arrest most probably due to acute thiamine deficiency (such sudden cardiac arrests were well known amongst prisoners-of-war in the Far East, and indeed wherever beriberi is endemic), and thirdly a coincidental bronchopneumonia in undernourished asthenic and neglected individuals.

The argument that alcoholic addiction may be replaced by paraldehyde (or other sedative) addiction is not pertinent to the treatment of acute alcoholic hallucinosis, which clears up within a few days. It is merely a confession of our failure to treat satisfactorily the underlying causes of alcoholism—a different and a much more difficult task.

One must agree entirely with Dr. Steyn's emphasis on the need to promptly treat the general state of malnutrition which is present in the majority of these patients, and as a guide to sound management commend the paper of E. G. Saint *et alii* (M. J. AUSTRALIA, May 31, 1952).

It is advisable to give all sufferers from alcoholic confusional or hallucinatory states repeated routine intravenous thiamine for the first forty-eight hours after admission in an attempt to avert the odd sudden beriberi death or Wernicke's syndrome.

Finally, although oral paraldehyde is completely safe, we have become increasingly disturbed at this hospital by the not infrequent sterile abscesses which follow intramuscular paraldehyde, and its use by this route is confined only to emergencies. It has never been given intravenously, and I see no reason why it ever should.

Yours, etc.,

JOHN F. J. CADE,
Psychiatrist Superintendent.

Mental Hospital and Receiving House,
Royal Park, N.2,
Victoria.
July 24, 1953.

SIR: With the exception of the cases reported by Hemphill and Heller, the reference to which is not given in Dr. Steyn's paper and does not appear in the "Index Medicus" for 1945, all the doses mentioned by him are much greater than those usually given. Any sedative may prove unsatisfactory in the control of restlessness in *delirium tremens*, and in my opinion it has yet to be shown that paraldehyde in doses of two or three drachms by mouth or ten cubic centimetres intramuscularly to begin with, is either useless or unsafe. The barbiturates, too, are toxic when the liver is damaged. The mistake which both Dr. Minogue (M. J. AUSTRALIA, 1952, 2: 254) and Dr. Steyn emphasize is to depend on sedation to the neglect of good nursing and adequate diet together with insulin in subcoma doses. Piker and Cohn (*J.A.M.A.*, 1937, 108: 345) claim to have reduced the mortality in *delirium tremens* by their intensive therapy including insulin and consider paraldehyde the safest hypnotic. They state: "That morphine increases the chances of death in a case of delirium tremens is not to be doubted." Noyes in "Modern Clinical Psychiatry" (1948, Philadelphia) states: "It is frequently desirable to give some of the less depressing hypnotics such as paraldehyde in 3 to 4 drachms doses once to three times a day. Morphine should never be given." Henderson and Gillespie (1950) state that paraldehyde is preferably avoided owing to its tendency to increase the restlessness and also refer to the sedative effect of insulin. They recommend hyoscine (one-hundredth to one-fiftieth of a grain) "as a last resort". The present writer has always looked on hyoscine as a most dangerous drug, never to be given alone, and has preferred to resort to electroplexy when reasonable doses of sedative are proving ineffective.

Yours, etc.,

W. S. DAWSON.

Sydney,
July 21, 1953.

EARLY DIAGNOSIS OF CERVICAL CARCINOMA WITH SPECIAL REFERENCE TO GENERAL PRACTICE.

SIR: May I congratulate Dr. R. Francis Matters on his article "Early Diagnosis of Cervical Carcinoma with Special Reference to General Practice" which appeared in the journal of June 20, 1953. As a matter of history, however, may I correct the statement that the Cancer Detection Clinics were introduced to Tasmania by Sir Edward Hallstrom. The clinics are largely maintained by Sir Edward, and they are known as Hallstrom Clinics. The credit for their introduction, however, must be given to the Minister for Health, Dr. R. J. David Turnbull.

Yours, etc.,

C. CRAIG,
Chairman, Tasmanian Cancer Committee.

Frederick Street,
Launceston,
July 20, 1953.

LEAD INTOXICATION IN THREE FAMILIES.

SIR: I should like to congratulate Dr. Bridge on her excellent article "Lead Intoxication in Three Families" in the journal of July 11. I should, however, like to point out that there was one other Australian report (Rischbieth, 1948) of familial incidence of this condition during the last five years, when two brothers were seen, one of whom was quite ill, the other virtually asymptomatic.

Yours, etc.,

HENRY RISCHBIETH, M.B., B.S., M.R.A.C.P., D.C.H.

63 Palmer Place,
North Adelaide,
South Australia.
July 23, 1953.

Reference.

RISCHBIETH, H. G. (1948), "Lead Poisoning", *Clin. Rep. Adelaide Child. Hosp.*, 1: 132.

CHILBLAINS.

SIR: In the special article for the clinician on chilblains appearing in the July 25 issue of the journal no mention is made of the effect of ultra-violet radiation. In our hands this has proved by far the most satisfactory and lasting treatment. The technique employed is to irradiatae the affected areas with the Kromayer lamp: initial dose four seconds, increasing by two seconds every three to four days. Symptoms are relieved by an average of four doses. Two to three prophylactic doses at the commencement of winter will usually keep the patient free for the ensuing cold weather.

Yours, etc.,

B. G. WADE.
T. W. BURGESS.

135 Macquarie Street,
Sydney,
July 29, 1953.

HEREDITY AND MENTAL DISORDER.

SIR: Dr. Cantor's finding (M. J. AUSTRALIA, July 25, 1953) that few of the mongols under his care have been the last of a large family, and that the parents in most cases have been young, suggests that his series was not a random selection. Penrose (*J. Ment. Sc.*, 1944, 90: 404) states: "It is becoming more widely accepted that two unknown factors, one associated with advancing maternal age rather than with birth order only, the other a genetic influence irregularly dominant with possibly a chromosome abnormality, are ætiologically significant." Over half of Penrose's cases were last born. The coexistence of mental deficiency and cyclothymic trends in the schizophrenic twins points to multiple genetic factors. Mott's law of anticipation appears to have been disposed of by Paterson (*J. Neurol. & Psychopath.*, 1933, 13: 193) and others.

Yours, etc.,

W. S. DAWSON.

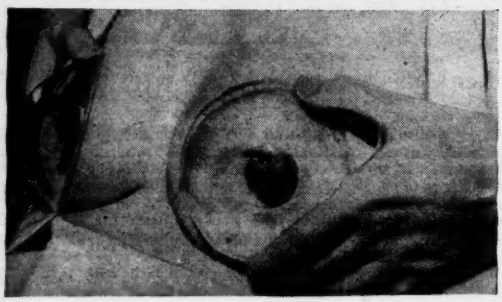
Sydney,
July 31, 1953.

ACUTE MASTITIS: A STUDY OF 28 CASES.

SIR: May I be allowed belatedly to congratulate Dr. Clair Isbister on her article on acute lactational lactitis which appeared in your journal of December 6, 1952? Her findings confirm clinical experience of the condition, its main incidence in *primiparæ*, its association with overloading of the breasts at the start of lactation, and their incomplete drainage.

Granted these, a breach of the nipple's surface, and staphylococcal infections in the nursery, and all the predisposing conditions for acute mastitis are fulfilled. Despite the help of antibiotics breast abscesses are more frequent than they should be, and my object in writing is to support Dr. Isbister's plea for greater vigilance in their prevention.

In her first series of 28 cases 27 of the women had sustained injury to a nipple; and in the second group at least 11 out of 17, with four whose records on this point were incomplete. This gives a total of 38, possibly 42, out of 45; a percentage of 84, possibly 93. This injury occurs during the early days of suckling when by reason of maldevelopment the nipple is anchored to the deep tissues of the breast. It



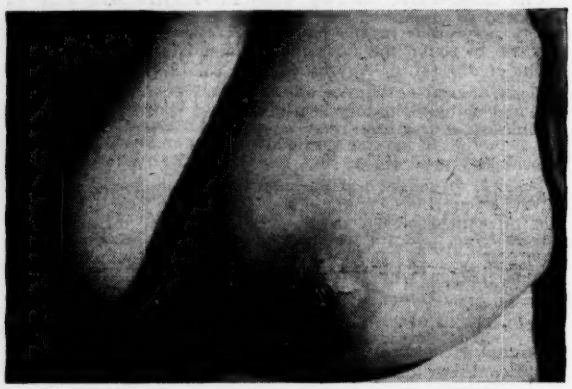
FIGURES IA AND IB.

The type of nipple shield recommended to stretch the attachment of the retracting nipple. It is worn under a well-fitting brassière. The shields, made of glass and also of a plastic material, are made and distributed by Allen and Hanbury, Bethnal Green, London, E.2.

cannot be drawn far into the baby's mouth, with the consequence that the main force of suction falls upon its surface instead of on the skin of the areola. If as many as six of 28 women had actually inverted nipples "which were not recognized or treated in pregnancy", it is unlikely that those which were merely anchored would receive attention; yet because they retract when the baby is suckled, they are just as likely to be damaged. It is so important that doctors and midwives should appreciate this that I venture to send an illustration of a very simple test which will always reveal the defect and takes but a moment to perform. Applied as a routine early in pregnancy as part of ante-natal examination, the retracting nipple can be corrected, or at least much improved, by the use of a shield, also illustrated, worn under a suitable brassière. This simple preparatory measure greatly reduces the risk of acute mastitis.

Nevertheless, damage will sometimes occur, and its correct management is important. As Dr. Isbister points out, the pain of suckling when the nipple's surface is broken is severe enough to inhibit the reflex ejection of milk; this in turn leads to stasis and favours an ascending infection. The midwife should be taught to accept a ruling that suckling must be withheld from the first experience of pain, and the breasts emptied by hand until the epithelium is securely healed. She should indeed go further and throughout the lying-in period regularly ask her patient whether suckling hurts. For many years at my hospital the nursing staff have been taught that every "cracked" nipple is a potential breast abscess.

May I add a few words on the treatment of acute mastitis? Dr. Isbister's account of its onset cannot be improved, but I would point out that the type that begins with a general feeling of illness, intense headache and shivering is often attributed to a "chill" or labelled "flu", and is treated with aspirin. Time is thus lost. If mastitis exists, careful examination will always discover a tender area somewhere in one or other breast, though at this stage the patient may make no mention of pain. It is now that penicillin given in large dosage can be almost relied upon to extinguish the infection within a matter of hours, despite the fact that the staphylococcus grown from the milk may subsequently be stated by the pathologist to be penicillin-resistant. Our custom is to prescribe 1,000,000 units and repeat this four hours later, continuing with half this amount once daily for the next forty-eight hours. It is of great advantage to give stilbœstrol at the same time, and if the breasts are secreting actively the dosage of this also had better be high—for example, 15, 10 and 10 milligrammes at four-hourly intervals. Reduction



FIGURES IIA AND IIB.

To illustrate the test recommended to reveal retraction of the nipple. The first finger and thumb are pushed into the breast at the margin of the areola and pressed together in imitation of the action of the baby's jaws. This nipple, though apparently well formed, retracts level with the surface of the breast. (By courtesy of the *Nursing Mirror*.)

of the milk yield is of no consequence, for it will be made good within a few days after suckling has been resumed.

Dr. Isbister does not give figures to show what sort of frequency her cases of breast abscess represent. At the British Hospital for Mothers and Babies in 1951 there were six cases of mastitis in the 1400 women delivered (0·4%). Treated in the way described no breast abscess occurred, and in none was breast-feeding interrupted for more than a few days. The need to recognize its connexion with infection in the newborn needs all the emphasis given it by your contributor. Skin sepsis and the sadly misnamed "sticky eye" are too often looked upon as trivialities. A breast abscess is a disaster which leaves a bad memory and often causes a mother to postpone indefinitely another pregnancy; or even, as I have more than once known, cause her to seek to have an abortion produced.

Yours, etc.,

HAROLD WALLER, F.R.C.O.G.
(British Hospital for Mothers and Babies, Woolwich.)
Tunbridge Wells, England.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Film on the Care of the Premature Baby.

THE Post-Graduate Committee in Medicine in the University of Sydney has had prepared a film entitled "Care of the Premature Baby". This was shown at a *première* in the Robert H. Todd Assembly Hall, 135 Macquarie Street, Sydney, on July 29, 1953.

The film shows in straightforward fashion the procedure advised to promote survival and healthy development of the premature baby, illustrating suitable means of providing for the child's needs in such things as warmth, satisfactory respiration, feeding, freedom from infection and minimal handling. Emphasis is laid on the importance of defining standards of prematurity and of adopting uniform methods of keeping records in hospitals. The film is in good colour and is based on a lecture script prepared by Dr. Kathleen Winning, who also acted as technical director. The script was submitted to each of the post-graduate committees in medicine in Melbourne, Adelaide and Brisbane for comments and suggestions by obstetricians and pædiatricians in their own States; but the final decision on material to be included rested with the Post-Graduate Committee in Sydney. The production of the film, by Kingcroft Productions, is excellent. The entire cost of the film was borne by Nicholas Proprietary, Limited.

"Care of the Premature Baby" has been added to the film collection of the Post-Graduate Committee, which is being temporarily housed and handled by the New South Wales Film Council. It is available for use in other States as well as in New South Wales. As with other films in the Post-Graduate Committee's collection, intending borrowers should first apply to the Committee for registration. They may then obtain this and other films direct from the New South Wales Film Council by complying with the Council's rules.

The Royal College of Obstetricians and Gynaecologists.

VICTORIAN STATE COMMITTEE.

Lecture-Discussions.

A COURSE of lecture-discussions of special interest to general practitioners will be given in the Pathology Lecture Theatre of the Women's Hospital, Swanston Street, Melbourne, as follows:

Wednesday, September 23, 1953, at 8.15 p.m.: "Puerperal Infection", A. M. Hill, F.R.C.O.G. Chairman: V. E. Hollyock, M.R.C.O.G.

Wednesday, October 14, 1953, at 8.15 p.m.: "Labour Ward Emergencies", R. M. Rome, M.R.C.O.G. Chairman: J. Smibert, M.R.C.O.G.

Wednesday, October 28, 1953, at 8.15 p.m.: "The Retroverted Uterus", F. J. Hayden, F.R.C.O.G. Chairman: J. G. O'Donoghue, M.R.C.O.G.

The fee for the course of three lecture-discussions is one guinea, or 10s. 6d. for individual lectures. Fees may be paid to the Secretary, Royal College of Obstetricians and Gynæcologists, Victorian State Committee, 122 Flinders Street, Melbourne, or on admission.

Congresses.

SECOND INTERNATIONAL CONGRESS OF CARDIOLOGY.

THE second International Congress of Cardiology will be held in Washington, D.C., from September 12 to 15, 1954. It will be immediately followed by the annual scientific sessions

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 11, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1(1)	3(2)	..	..	..	..	..	..	4
Amœbiasis		1(1)	..	..	..	..	..	..	1
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	1	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	4(4)	20(19)	..	..	..	..	..	24
Diphtheria	13(7)	3(2)	8(5)	..	2(2)	..	..	..	26
Dysentery (Bacillary)	..	8(8)	1(1)	..	..	1(1)	..	..	10
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	11(4)	..	..	19(14)	..	..	..	30
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	2(1)	..	..	..	..	..	2
Malaria	..	..	..	..	2(2)	..	..	..	2
Meningococcal Infection	3(2)	1	2(2)	..	..	1(1)	..	..	7
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	2	..	..	..	..	..	..	..	2
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	5(3)	1(1)	1	5(3)	2(2)	1	..	..	15
Puerperal Fever	..	1	1(1)	..	..	..	..	..	2
Rubella	..	30(13)	..	..	17(12)	..	..	..	47
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	13(5)	67(35)	2	5(3)	..	1	..	..	88
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	1	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	32(28)	17(11)	6(4)	2(2)	6(6)	7(4)	..	..	70
Typhoid Fever	1(1)	3(3)	..	..	..	1(1)	..	..	5
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

of the American Heart Association, from September 16 to 18, 1954. The scientific sessions lasting for three days will include formal papers, panel discussions, clinical pathological conferences and visits to important medical centres in Washington and Bethesda. A series of post-congressional visits and conferences to at least 20 of the leading cardiac clinics in different parts of the United States of America and Canada has been arranged by special committees of local Heart Associations in the various cities. Correspondence relating to the Congress should be addressed to L. W. Gorham, M.D., Secretary-General, Office of the Second International Congress of Cardiology, 44 East 23rd Street, New York 10, New York, United States of America.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 46, of July 23, 1952.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Volunteer Reserve.

Promotion.—Surgeon Lieutenant (Surgeon Lieutenant-Commander (Acting)) John Francis Rutter is promoted to the rank of Surgeon Lieutenant-Commander, dated 7th October, 1950.

Resignation.—The resignation of Franklin Robert Fay of his appointment as Surgeon Lieutenant-Commander is accepted, dated 31st March, 1953.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The probationary appointment of the following Flight Lieutenants is confirmed: J. McC. Morrison (036133), R. G. Hall (024849), R. W. L. Hall (024941), F. J. G. Arnold (025085), M. A. May (013705), G. G. Wagner (013706), N. L. Groves (025211), D. M. Newman (013992), W. E. Downey (036102).

Medical Practice.

POLICE OFFENCES (AMENDMENT) ACT, 1908, NEW SOUTH WALES, AS AMENDED: REGULATION.

THE following amendment to the drug regulations under the *Police Offences (Amendment) Act*, 1908, as amended, is published in the *New South Wales Government Gazette* of July 17, 1953. The amendment concerns the basis for withdrawal of a drug authority.

Regulation 25 is amended by *inserting* in paragraph (1) after the word "Regulations" where secondly occurring the words "or has been charged with any offence against the Act or the Regulations and in respect of such charge an order has been made under subsection one of Section 556A of the Crimes Act, 1900, as amended by subsequent Acts".

Australian Medical Board Proceedings.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners: Horne, Terence William, M.B., B.S., 1948 (Univ. Sydney); Ferguson, Margaret, M.B., B.S., 1949 (Univ. Melbourne); Were, Stuart Owen Mansel, M.B., B.S., 1946 (Univ. Melbourne), D.P.M. and H. (Univ. Sydney), 1953; McKay, Stanley Robert, M.B., B.S., 1952 (Univ. Melbourne).

Deaths.

THE following death has been announced:

SCHUETTE.—Rudolph William Louis Schuette, on August 1, 1953, at Springwood, New South Wales.

Diary for the Month.

AUG. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
AUG. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 19.—Western Australian Branch, B.M.A.: General Meeting.
AUG. 19.—Victorian Branch, B.M.A.: Clinical Meeting.
AUG. 20.—Victorian Branch, B.M.A.: Executive of Branch Council.
AUG. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 22.—Queensland Branch, B.M.A.: Annual General Meeting.
AUG. 25.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 26.—Victorian Branch, B.M.A.: Branch Council.
AUG. 27.—New South Wales Branch, B.M.A.: Branch Meeting.
AUG. 28.—Queensland Branch, B.M.A.: Bancroft Oration.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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